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Beverage consumption and risk of ulcerative colitis

Systematic review and meta-analysis of epidemiological studies

Jia-Yan Nie, MD[a,b], Qiu Zhao, MD[a,b,*]

Abstract

Epidemiological studies have provided controversial evidence between beverage consumption and the risk of ulcerative colitis (UC). This study aimed to determine the role of beverage consumption in the development of UC. A systematic search was conducted in public databases to identify all relevant studies, and study-specific relative risks (RRs) and 95% confidence intervals (CIs) were pooled using a random-effects model. Sixteen studies were identified with a total of 3689 cases and 335,339 controls. Alcohol consumption showed no significant association with UC risk (RR for the highest vs the lowest consumption level: 0.95, 95% CI: 0.65–1.39). Coffee consumption tended to be inversely associated with UC risk (RR: 0.58, 95% CI: 0.33–1.05), but it was not significant and confounded by smoking adjustment. Soft drinks consumption was associated with UC risk (RR: 1.69, 95% CI: 1.24–2.30), and tea consumption was inversely associated with UC risk (RR: 0.69, 95% CI: 0.58–0.83). In conclusion, high consumption of soft drinks might increase the risk of UC, while tea consumption might decrease the risk.

Abbreviations: FFQ = food frequency questionnaire, HR = hazard ratio, IBD = inflammatory bowel disease, NOS = the Newcastle-Ottawa Scale, OR = odds ratio, RR = relative risk, UC = ulcerative colitis.

Keywords: alcohol, coffee, meta-analysis, soft drinks, tea, ulcerative colitis

1. Introduction

Ulcerative colitis (UC) is a chronic inflammatory disorder of the intestinal tract, which is clinical characterized by bloody diarrhea, abdominal pain and extraintestinal manifestations.[1] The incidence was steadily on the rise globally during the past 2 decades.[2] As for the frequent relapses, poor efficacy of meditation and high risk of surgery,[3,4] the UC patients are heavily burdened with low quality of life and medical costs.[5,6] However, the etiology was still unknown. There was growing evidence that dietary factors played a role in the development of UC.[7] In the meta-analysis of Li et al,[8] consumption of vegetables and fruit were identified to be inversely associated with the risk of UC (OR: 0.71, 95 CI%: 0.58–0.88; OR: 0.69, 95 CI%: 0.49–0.96). In the meta-analysis of Wang et al,[9] dietary sucrose intake was also positively related with UC risk (RR for per 10 g increment/day: 1.098, 95% CI: 1.024–1.177).

During the past decades, the prevalence of western diet coincided with an increasing incidence of UC in those regions with a primary low incidence. Thus, western diet was usually regarded as a risk factor for UC.[10,11] As a key feature of western diet, beverage consumption might also play some role in the development of UC. However, previous epidemiological studies reached inconsistent results, and no meta-analyses have focused on this. Therefore, we conducted a systematic review and meta-analysis to determine the role of beverage consumption in the development of UC.

2. Materials and methods

2.1. Search strategy

The databases of PubMed, Embase, and Cochrane Library databases were searched for relevant studies published up to August 1, 2017, using the key words "beverage," "tea," "alcohol," "wine," "beer," "liquor," "coffee," "soda," "soft drinks," "diet," "environmental factor," "risk factor" in combination with "inflammatory bowel disease," "ulcerative colitis," "IBD," and "UC." Moreover, we also reviewed the reference lists of related studies, reviews, and meta-analyses for undetected studies. This study was approved by the ethics committee of Zhongnan Hospital of Wuhan University.

2.2. Study selection exclusion

All the studies were reviewed independently by 2 investigators (J-YN and QZ). Studies were included if they satisfied the following criteria: observational studies published originally; measured the consumption levels of at least one of the beverages (tea, alcohol, coffee, and soft drinks) by food frequency questionnaires (FFQs)

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which contained various food items and related consumption frequency; the diagnosis of UC by clinical symptoms, endoscopy and histology; evaluated the association between beverage consumption and UC risk; presented relative risks (RRs), odds ratios (ORs), hazard ratios (HRs) with 95% confidence intervals (CIs). We excluded abstracts without full texts and review articles.

2.3. Data extraction and quality assessment

The following information was extracted from each included study: authors, publication year, area, study design, number of cases and controls, beverage types, exposure assessment, estimates, and adjustors.

The Newcastle-Ottawa Scale (NOS), which contained 9 terms with each term accounting for 1 score, was used to assess the methodological quality of included studies.[12]

2.4. Statistical analysis

As the absolute risk of UC is low, OR and HR were roughly regarded as RR in this meta-analysis.[13] To evaluate the risk of high consumption of beverages, we pooled the risk estimates for the highest versus the lowest consumption level. A random-effects model was used as the pooling method, which considers both within-study and between-study variation. The heterogeneity between studies was estimated by Q test and I^2 statistic, and $I^2 > 50\%$ represented substantial heterogeneity.[14] Subgroup analysis was performed on study design, ethnicity, and smoking adjustment to investigate the stability of main results. In addition, sensitivity analysis was conducted by deleting each study in turn to reflect the influence of individual data on the pooled results. Egger test was used to detect publication bias.[15] If publication bias was present, the "trim and fill" strategy was used to adjust the funnel plot and then recomputed the results.[16] All statistical analyses were performed using Stata SE12.0 software (StataCorp LP, College Station, TX), and all tests were sided with a significance level of .05.

3. Results

3.1. Characteristics of included studies

The search strategy identified 11,499 records: 2718 from PubMed, 8755 from Web of Science, and 26 from other sources (Fig. 1). After eliminating duplicated and irrelevant records, 16 studies were included in the meta-analysis (Table 1).[17–32] Among the 16 studies, there were 13 case–control and 3 prospective cohort studies, with a total of 3689 cases and 335,339 controls. In study quality assessment, the quality scores ranged from 6 to 8, with an average of 6.38 (Table S1, http://links.lww.com/MD/B1000; Table S2, http://links.lww.com/MD/B1000).

3.2. Alcohol consumption and UC risk

Nine studies evaluated the association between alcohol consumption and UC risk. The summary RR for the highest versus the lowest intake was 0.95 (95% CI: 0.65–1.39, $I^2 = 66.9\%$, $P_{\text{heterogeneity}} = .002$), indicating no obvious association between them (Fig. 2). Sensitivity analysis showed the result was robust (Fig. S1, http://links.lww.com/MD/B1000). Egger test detected significant publication bias ($P = .030$) (Fig. S2, http://links.lww.com/MD/B1000). After introducing the "trim and fill" method to adjust this bias, the overall estimate was still not significant (RR: 1.08, 95% CI: 0.66–1.51). In subgroup analysis, no substantial changes of the primary result were found between subgroups (Table 2).

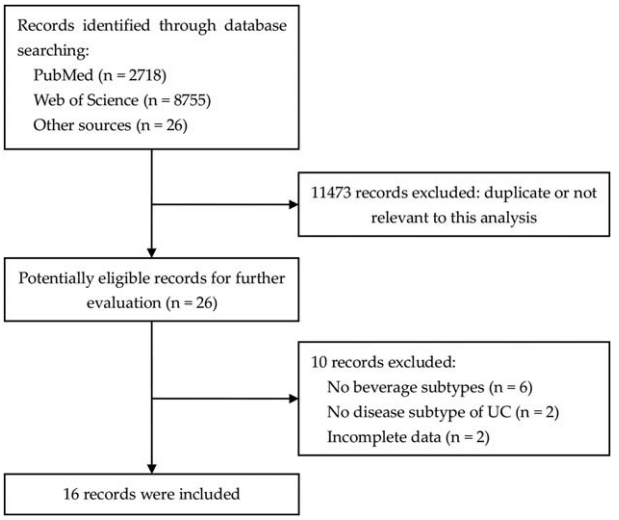

Figure 1. Flowchart of literature search.

3.3. Coffee consumption and UC risk

Six studies evaluated the association between coffee consumption and UC risk. The pooled RR for the highest versus the lowest intake was 0.58 (95% CI: 0.33–1.05, $I^2 = 87.5\%$, $P_{\text{heterogeneity}} < .001$), suggesting a potential but not significant role of coffee consumption in the development of UC (Fig. 2). In sensitivity analysis, the estimates became significant when omitting the studies by Russel et al (Fig. S3, http://links.lww.com/MD/B1000). In subgroup analysis, coffee consumption showed an inverse association with UC risk when not adjusted by smoking (RR: 0.41, 95% CI: 0.22–0.74) (Table 2). Egger test detected no significant publication bias ($P = .566$) (Fig. S4, http://links.lww.com/MD/B1000).

3.4. Soft drinks consumption and UC risk

Five studies evaluated the association between soft drinks consumption and UC risk, among which one specialized in the subtype of cola drinks. The pooled RR for the highest versus the lowest intake was 1.69 (95% CI: 1.24–2.30, $I^2 = 12.9\%$, $P_{\text{heterogeneity}} = .332$) (Fig. 2). Sensitivity analysis showed the result was robust (Fig. S5, http://links.lww.com/MD/B1000). In subgroup analysis, no substantial changes of the primary result were found between subgroups (Table 2). Egger test detected no significant publication bias ($P = .349$) (Fig. S6, http://links.lww.com/MD/B1000).

3.5. Tea consumption and UC risk

Three studies evaluated the association between tea consumption and UC risk. The pooled RR for the highest versus the lowest intake was 0.69 (95% CI: 0.58–0.83, $I^2 = 0.0\%$, $P_{\text{heterogeneity}} = .697$) (Fig. 2). Sensitivity analysis showed the result was robust (Fig. S7, http://links.lww.com/MD/B1000). In subgroup analysis, no substantial changes of the primary result were found between

Table 1

Characteristics of included studies.

Study	Area	Design	Cases/controls	Type	Consumption	Estimates (95% CI)	Adjustors
Boyko et al[17]	America	Population-based case–control	209/209	Coffee	≥6 cups/day vs none	1.4 (0.6–3.6)	Age, sex, smoking, income, educational level, occupational status
				Alcohol	≥5 cups/day vs none	0.4 (0.2–1.0)	
Persson et al[18]	Sweden	Population-based case–control	145/305	Coffee	≥3 cups/day vs none	0.3 (0.2–0.5)	Age, sex
Kono et al[19]	Japan	Hospital-based case–control	101/143	Soft drinks	High vs low	1.1 (0.5–2.2)	Age, sex, study area, inpatient status
				Alcohol	Current ≥15 mL/day vs never	0.9 (0.3–2.5)	Age, sex, study area, inpatient status, either smoking or alcohol use
Reif et al[20]	Israel	Population/hospital-based case–control	54/85	Soft drinks	High vs low	3.39 (1.25–9.19)	Age, sex, ethnic origin, area of residence, energy intake
Russel et al[21]	The Netherlands	Population-based case–control	398/616	Cola drinks	>1 vs 0–1 times/week	1.6 (1.1–2.3)	Age, sex, smoking, educational level, selected nutritional factors
				Coffee	≥1 times/week vs none	1.2 (0.9–1.7)	
Sakamoto et al[22]	Japan	Hospital-based case–control	108/211	Alcohol	Quartile 4 vs quartile 1	0.64 (0.27–1.55)	Age, sex, study area, education, smoking, energy intake
Halfvarson et al[23]	Sweden/Denmark	Population-based case–control	250/250	Coffee	≥3 cups/day vs none	0.10 (0.03–0.40)	Di- and monozygotic pairs
Jiang et al[24]	China	Population-based case–control	177/177	Alcohol	Frequent vs none/rare	0.91 (0.46–1.83)	Age, sex
				Tea	Heavy vs none/rare	0.65 (0.34–1.26)	Age, sex, smoking, drinking, capsicum consumption, appendectomy, family history
Hart et al[25]	European	Prospective	139/556	Alcohol	Quartile 4 vs quartile 1	0.84 (0.46–1.52)	Age, sex, study center, recruitment date, total energy intake, smoking
Hansen et al[26]	Denmark	Hospital-based case–control	144/144	Coffee	≥3 vs <3 cups/day	0.89 (0.52–1.53)	Age, sex, ethnicity, geographic location
Jakobsen et al[27]	Denmark	Population-based case–control	56/477	Soft drinks	≥4 vs ≤3 times/week	3.3 (1.0–10.1)	—
Wang et al[28]	China	Population-based case–control	1308/1308	Tea	Heavy vs none/rare	0.738 (0.591–0.922)	Age, sex, study area
				Alcohol	Heavy vs none or rare	1.453 (1.122–1.882)	
Ng et al[29]*	Asia-Pacific	Population-based case–control	256/940	Soft drinks	≥2 times/week vs not	1.553 (0.831–2.899)	Age, sex, geographical location, country income
				Coffee	Daily vs not	0.490 (0.350–0.686)	
				Tea	Daily vs not	0.63 (0.465–0.853)	
Hsu et al[30]	China	Population-based case–control	97/287875	Alcohol	Alcohol intoxication vs none	2.33 (1.39–3.90)	Age, sex, comorbidity
Bergmann et al[31]	Europe	Prospective	198/792	Alcohol	Heavy vs light	0.95 (0.52–1.76)	Age, sex, center, enrolment date, educational attainment, smoking
Porter et al[32]	America	Prospective	49/41251	Alcohol	Heavy vs no/light	0.16 (0.02–1.19)	Age, antecedent infectious gastroenteritis, marital status, service branch, occupation, smoking, life stressors

CI = confidence interval.

*Also included the analysis for Asians only.

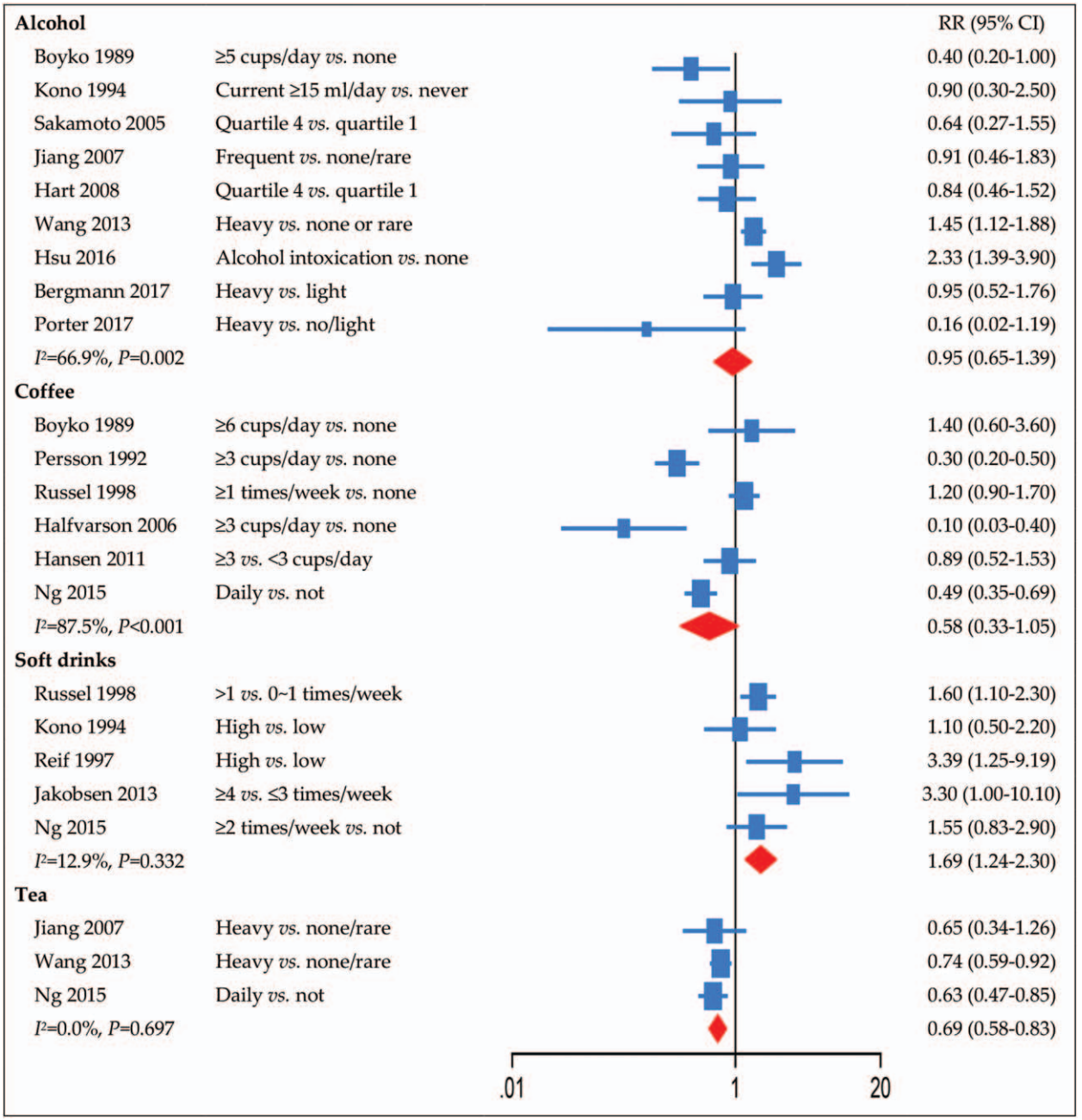

Figure 2. Forest plot of beverage consumption and risk of ulcerative colitis.

subgroups (Table 2). Egger test detected no significant publication bias ($P = .623$) (Fig. S8, http://links.lww.com/MD/B1000).

4. Discussion

This is the first meta-analysis to study the association between beverage consumption and UC risk, and 4 main daily subtypes were analyzed respectively. For alcohol consumption, it was not associated with UC risk (RR: 0.95, 95% CI: 0.65–1.39). However, alcohol could cause direct mucosal injury and increase bacterial translocation, and it was usually regarded as a risk factor for UC.[33] Our study suggested an insignificant role of alcohol consumption in the development of UC. We thought the inconsistency contributed to the difference between experimental studies and epidemiological studies, and the latter considered more confounders. Just like fat and the subtypes, it was positively or inversely associated with experimental colitis, but epidemiological studies indicated an insignificant association with UC risk.[34]

For coffee consumption, it tended to be inversely associated with UC risk (RR: 0.58, 95% CI: 0.33–1.05), but not significant. In vitro, caffeine treatment in intestinal epithelial cell lines could reduce bacterial invasion though downregulating CHI3L1.[35] In vivo, mice treated with caffeine displayed a delayed response toward dextran sulfate sodium-induced colitis. These indicated a protective role of coffee consumption in UC. We thought the inconsistency with our study might contribute to the limited number of included studies, or the protective effects might be

Table 2

Subgroup analysis of beverage consumption and risk of ulcerative colitis.

	Alcohol			Coffee			Soft drinks			Tea		
Subgroup	Num	RR (95% CI)	I^2	Num	RR (95% CI)	I^2	Num	RR (95% CI)	I^2	Num	RR (95% CI)	I^2
Study design												
Hospital-based	2	0.73 (0.37–1.44)	0.0	1	0.89 (0.52–1.53)	—	1	1.10 (0.52–2.31)	—	—	—	—
Population–based	7	0.99 (0.64–1.53)	72.4	5	0.53 (0.26–1.06)	89.7	3	1.67 (1.23–2.27)	0.0	3	0.69 (0.58–0.83)	0.0
Ethnicity												
Asian	5	1.27 (0.85–1.90)	55.9	1	0.51 (0.36–0.72)	—	1	1.43 (0.74–2.76)	—	3	0.70 (0.59–0.83)	0.0
Caucasian	4	0.66 (0.39–1.11)	42.1	5	0.59 (0.28–1.27)	88.7	4	1.80 (1.15–2.81)	33.8	—	—	—
Smoking												
Yes	6	0.71 (0.50–1.01)	7.8	2	1.22 (0.90–1.65)	0.0	1	1.60 (1.11–2.31)	—	1	0.65 (0.34–1.25)	—
No	3	1.51 (0.99–2.30)	58.9	4	0.41 (0.22–0.74)	79.4	4	1.84 (1.10–3.07)	33.2	2	0.70 (0.58–0.84)	0.0

CI = confidence interval, Num = number of included studies, RR = relative risk.

weakened by other risk factors especially when not all potential confounders were adjusted in the included studies. Moreover, the result was confounded by smoking adjustment, which has been validated in an inverse association with UC risk.[36] Large-scale prospective studies were warranted to further investigate the association.

For the consumption of soft drinks, it was associated with UC risk (RR: 1.69, 95% CI: 1.24–2.30). Soft drinks had been a highly visible and controversial public health issue, which were also viewed by many as a major contributor to obesity and related chronic diseases.[37,38] In the prospective study of Racine et al,[39] there also found a positive association between a "high sugar and soft drinks" pattern and UC risk.

For tea consumption, it had a reverse association with UC (RR: 0.69, 95% CI: 0.58–0.83). Animal studies found that tea alone and in combination with sulfasalazine could inflammatory changes in experimental colitis, indicating a protective role of tea in UC.[40,41]

This meta-analysis had several strengths. First, this is the first meta-analysis to study the association between beverage consumption and UC risk. Second, we evaluated 4 most daily subtypes (alcohol, coffee, soft drinks, and tea). There were also several limitations. First, the results based on case–control studies were prone to introduce considerable bias, particularly recall bias and interviewer bias. Second, there existed considerable amount of heterogeneity in the meta-analyses of alcohol and coffee. We thought it might contribute to the limited number of included studies and other potential confounders (e.g., differences in genetic background, beverage types, consumption quantification, and grouping cutoff). Thus, sensitivity and subgroup analyses were used to test the stability of the results, and the results were stable in general. Third, not all potential confounders were adjusted in every study.

5. Conclusions

In conclusion, high consumption of soft drinks might increase the risk of UC, while tea consumption might decrease the risk.

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