Cancer Causes Control (2010) 21:1843–1850
DOI 10.1007/s10552-010-9611-9

ORIGINAL PAPER

A meta-analysis of alcohol intake and risk of bladder cancer

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Received: 26 February 2010 / Accepted: 25 June 2010 / Published online: 9 July 2010

Abstract
Objective Epidemiologic studies have reported conflicting results relating alcohol intake to bladder cancer risk. A meta-analysis of cohort and case–control studies was conducted to pool the risk estimates of the association between alcohol intake and bladder cancer.
Methods Eligible studies were retrieved via both computer searches and review of references. We analyzed abstracted data with random effects models to obtain the summary risk estimates. Dose–response meta-analysis was performed for studies reporting categorical risk estimates for a series of exposure levels.
Results Nineteen studies met the inclusion criteria of the meta-analysis. No association with bladder cancer was observed in either overall alcohol intake group (OR = 1.00, 95% CI 0.89–1.10) or subgroups stratified by sex, study design, geographical region, or smoking status. However, in the analysis by specific beverages, both beer (OR = 0.86, 95% CI 0.76–0.96) and wine (OR = 0.85, 95% CI 0.71–1.00) consumption exhibited a negative dose–response relationship with bladder cancer.
Conclusion The overall current literature on alcohol consumption and the risk of bladder cancer suggested no association, while the consumption of beer and wine was associated with reduced risk of bladder cancer. Further efforts should be made to confirm these findings and clarify the underlying biological mechanisms.

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Keywords Alcohol drinking · Alcoholic beverages · Bladder neoplasms · Meta-analysis

Introduction

Alcohol is a major correlate of health and disease and is estimated to account for 3.6% of all cancer cases and 6% of cancer death worldwide [1, 2]. Although alcohol is not known to be carcinogenic in animal experimentation, considerable evidence from epidemiologic studies suggests that increased alcohol is a risk factor for cancers of the upper alimentary tract, liver, colorectum, and female breast [3]. Modifying alcohol consumption could be part of a prevention strategy of cancer through lifestyle changes.

Bladder cancer is the fourth most common cancer in the USA, with an expected 71,000 newly diagnosed cases and 14,300 deaths [4] in 2009. Many epidemiologic studies support a positive association between bladder cancer and chemical or environmental exposures, such as cigarette smoking, aromatic amines and *Schistosoma haematobium* infestation [5]. However, the findings on the association between alcohol intake and the risk of bladder cancer are inconsistent. A meta-analysis conducted in 1999 suggested a weak elevated but insignificant risk of urinary tract cancer from alcohol consumption for men [6], while the risk for women and the influence of the amount and type of alcohol remained unclear because of limited data. There have been other reviews of this association, but they have not combined risk estimates for an overall estimate of the effect size [7, 8].

The purpose of the present study was to update and quantitatively assess the association between alcohol consumption and the risk of bladder cancer by summarizing the results of published cohort and case–control studies.

We also sought to address the unresolved issue of whether this relationship differs across the specific alcoholic beverages.

Materials and methods

Literature research

We identified publications in MEDLINE database using PubMed, Web of Science and the Cochrane Library. "Alcohol" and "bladder cancer" were used as keywords in the free text words. The articles evaluating the relationship of urinary tract cancer and alcohol consumption were also retrieved, because the overwhelming majority of tumors occurred in the bladder, and the renal pelvis and ureter are covered by the same urothelium. The term bladder cancer was used as a synonym for these neoplasms. Additionally, we performed hand searches via cited references in the abstracted articles and previous reviews on alcohol and cancer. Each identified publication was reviewed and included in the analysis if all the following criteria were met: (1) case–control or cohort study published as an original article; (2) papers reported in English between 1980 and 2009 December; (3) findings expressed as odds ratio (OR) or relative risk (RR) and its 95% confidence intervals (95% CI) adjusted for at least age, sex and smoking, or sufficient information allowing us to compute them.

Data extraction

We extracted the name of the first author, the year of publication, the country in which the study was conducted, study design, sample size, anatomical site of the neoplasm, adjusted effects estimates, exposure assessment and adjusted covariates. Considering that bladder cancer is a rare disease, the RR was assumed approximately the same as OR, and the OR was used as the study outcome. Adjusted ORs were extracted directly from the original reports. If studies reported sex-stratified age- and smoking-adjusted ORs, we calculated the overall age-, smoking- and sex-adjusted OR by combining these estimates with the method of Mantel and Haenszel [9]. For studies that reported separate adjusted odds ratios for several consumption strata, we estimated the exposed versus non-exposed adjusted OR using the exposure-specific prevalence of the non-cases as weight [10].

Statistical analysis

We pooled data using the DerSimonian and Laird [11] random effects models, which consider both within-study and between-study variation. Subgroup analyses were performed according to sex (male or female), study design (cohort or case–control studies), the study location (USA/Canada, Europe or Asia), smoking status (non-smokers and smokers) and type of alcohol beverages (beer, wine, or spirits).

For the dose–response meta-analysis, we included studies considering at least 3 levels of alcohol consumption and providing the number of case patients and control subjects in each exposure category. We used the method proposed by Greenland [12] and Orsini [13] to back-calculate and pool the risk estimates. We converted all measures into gram alcohol per day on the widely used estimation that a standard drink contains 12 g of alcohol regardless of alcohol type unless it was defined in the study population or the geographical area. We assigned the level of alcohol consumption from each study to these categories based on the calculated midpoint of alcohol consumption. When the highest category was open-ended, we assumed the width of the interval to be the same as in the preceding category.

We quantified the extent of heterogeneity using Q test [11] and I^2 score [14] and statistical significance was considered while $P < 0.05$. Meta-regression analysis was used to explore the influence of study design, geographical region, alcohol assessment, and publication years in the heterogeneity. Publication bias was assessed using the tests of Egger [15] and Begg [16]. All statistical analyses were done with Stata Statistical Software, version 10.0.

Results

We identified 35 articles that examined the risk of bladder cancer with alcohol consumption published between 1980 and 2009 December. Upon closer examination, sixteen articles did not provide sufficient information to estimate a summary odds ratio and its 95% confidence intervals [17–22], or a summary odds ratio adjusted for at least age, sex and smoking [23–32]. The remaining 19 articles [33–51] were chosen for detailed review. Of the selected studies, six were cohort [38, 42, 43, 45, 50, 51], six were hospital-based case–control [34, 39, 41, 44, 46, 49], and seven population-based case–control studies [33, 35–37, 40, 47, 48] (Table 1). Eleven of these studies were conducted in the United States/Canada [34–38, 40, 45, 47, 48], while six were in Europe [33, 39, 41–44] and two in Asia [46, 49]. Thirteen articles reported the associations between consumption of specific alcoholic beverages (beer, wine or spirits) and the risk of bladder cancer [37, 38, 40–45, 47–51]. Information on alcohol consumption was obtained by interview, self-administered questionnaire or both techniques.

Among six cohort studies, five reported no significant association in drinkers as compared with nondrinkers [38,

Table 1 Study characteristics of published cohort and case–control studies on alcohol intake and bladder cancer

References	Study design	Country	Sex	Cases/ subjects	Anatomical site of urinary tract	Specific alcohol beverages	Variables of adjustment	Alcohol assessment
Mommsen et al. [33]	Population-based case–control study	Denmark	M/W	212/471	Bladder	No	Age, sex, smoking	Interview
Brownson et al. [34]	Hospital-based case–control study	USA	M	607/2,480	Bladder	No	Age, smoking	Questionnaire
Slattery et al. [35]	Population-based case–control study	USA	M/W	415/1,298	Bladder	No	Age, sex, smoking status, diabetes and bladder infection	Interview
Risch et al. [36]	Population-based case–control study	Canada	M/W	876/1,668	Bladder	No	Age, sex, residence, and lifetime cigarette consumption	Interview
Nomura et al. [37]	Population-based case–control study	USA	M/W	261/783	Lower urinary tract	Beer, wine, spirits	Age, sex, pack-years of cigarette smoking	Interview
Mills et al. [50]	Cohort	USA	M/W	52/34,198	Bladder	No	Age, sex, smoking, coffee, residence, sweetened fruit juice, vegetables, and meat	Questionnaire
Chyou et al. [38]	Cohort	USA	M	95/7,090	Lower urinary tract	Beer, wine, spirits	Age, smoking	Both techniques
Bruemmer et al. [40]	Population-based case–control study	USA	M/W	262/667	Bladder	Beer, wine, spirits	Age, sex, smoking, county	Interview
Donato et al. [39]	Hospital-based case–control study	Italy	M/W	172/750	Bladder	No	Age, sex, smoking, residence, education, date of interview, coffee	Interview
Pohlabeln et al. [41]	Hospital-based case–control study	Germany	M/W	300/600	Urinary tract	Beer, wine, spirits	Age, sex, smoking	Interview
Michaud et al. [51]	Cohort	USA	M	252/47,909	Bladder	Beer, wine, spirits	Geographic region, age, pack-years of smoking, current smoking status, energy intake, intake of fruits and vegetables, and intake of all other beverages	Questionnaire
Zeegers et al. [42]	Cohort	The Netherlands	M/W	594/3,764	Urinary tract	Beer, wine, spirits	Age, sex, smoking status, amount, and duration	Questionnaire
Geoffroy-Perez et al. [43]	Cohort	France	M/W	708/1,429	Bladder	Beer, wine	Age, sex smoking, residence, center	Interview
Pelucchi et al. [44]	Hospital-based case–control study	Italy	M/W	727/1,794	Bladder	Beer, wine, spirits	Age, sex, study center, education, smoking habits, coffee, tea, green vegetable intake, exposure to occupation at risk	Interview
Djousse et al. [45]	Cohort	USA	M/W	122/736	Bladder	Beer, wine, spirits	Age, sex, cohort, smoking status, and pack-years of cigarette smoking	Interview
Wakai et al. [46]	Hospital-based case–control study	Japan	M/W	124/744	Urinary tract	Beer, wine, spirits	Age, sex, cumulative consumption of cigarettes, year of first visit	Questionnaire

Table 1 continued

References	Study design	Country	Sex	Cases/ subjects	Anatomical site of urinary tract	Specific alcohol beverages	Variables of adjustment	Alcohol assessment
Jiang et al. [47]	Population-based case–control study	USA	M/W	1586/3,172	Bladder	Beer, wine, spirits	Age, sex, race, level of education, use of NSAIDs, carotenoid intake, number of years as hairdresser/barber, cigarette smoking status, duration of smoking, and intensity of smoking	Interview
Benedetti et al. [48]	Population-based case–control study	Canada	M	425/932	Bladder	Beer, wine, spirits	Smoking status, cigarette-years, time since quitting, respondent status, education, census tract income, ethnicity, age, fruit and vegetable consumption	Interview
Hemelt et al. [49]	Hospital-based case–control study	China	M/W	509/478 (beer) 486/499 (wine) 499/499 (spirits)	Bladder	Beer, wine, spirits	Age, sex, smoking status, smoking frequency, and smoking duration adjusted odds ratios	Interview

43, 45, 50, 51], whereas one study found an increased risk for men but not in women [42]. Most case–control studies found no significant association between alcohol drinking and bladder cancer, whereas two studies reported significantly increased risks [33, 39], and one found inverse associations [47]. A study conducted by Hemelt et al. [49] provided odds ratios for three alcohol beverages but no data on overall alcohol intake.

Figure 1 plots the pooled risk estimates for overall alcohol intake by study design. When all these studies were analyzed together, we observed alcohol intake was not associated with bladder cancer risk (OR = 1.00, 95% CI 0.89–1.10) and the summary ORs were similar across study design and source of the controls in case–control studies. There was no evidence of heterogeneity in cohort and hospital-based case–control studies, but some evidence in population-based case–control studies.

In Table 2, we pooled the OR estimates by sex (men and women), geographical region (USA/Canada, Europe and Asia), and smoking status (smokers and non-smokers). The OR estimates from subgroup analysis varied little, showing alcohol consumption was not associated with the likelihood of bladder cancer when separately analyzed by sex, geographical regions or smoking status. In analyses by specific beverages (Fig. 2), we found a significantly decreased risk of bladder cancer for intake of beer (OR = 0.86, 95% CI 0.76–0.96) and wine (OR = 0.85, 95% CI 0.71–1.00), but no association with spirits (OR = 1.01, 95% CI 0.87–1.15). Most of the results are

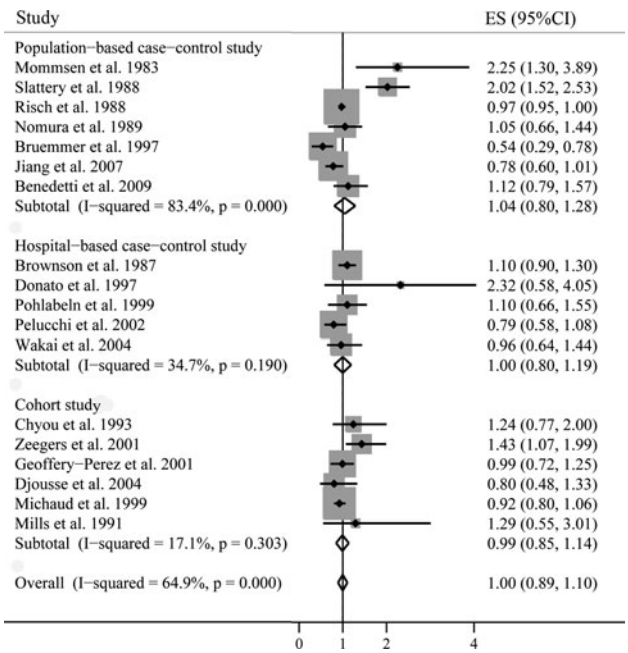

Fig. 1 A forest plot showing risk estimates from case–control and cohort studies estimating the association between overall alcohol consumption and risk for bladder cancer

Table 2 Summary of pooled odds ratios of bladder cancer by sex, geographical region, and type of alcohol beverages

Subgroup	Number of studies	Pooled OR (95% CI)	Q-test for heterogeneity *P* value (I^2 score)	Egger's test *P* value	Begg's test *P* value
All studies	18 [33–48]	1.00 (0.89, 1.10)	<0.001 (64.9%)	0.19	0.336
Sex					
Men	10 [34, 36, 37, 39–41, 43, 47, 48]	0.96 (0.83, 1.08)	<0.001 (72.4%)	0.71	0.15
Women	8 [36, 37, 39–41, 43, 44, 47]	0.9 (0.6, 1.21)	0.017 (59.1%)	0.74	0.17
Geographical region					
Asia	1 [46]	0.96 (0.64, 1.44)	–	–	–
Europe	6 [33, 39, 41–44]	1.13 (0.84, 1.42)	0.034 (58.6%)	0.17	0.35
USA/Canada	11 [34–38, 40, 45, 47, 48, 50, 51]	0.98 (0.85, 1.11)	<0.001 (72.4%)	0.59	0.94
Smoking status					
Non-smoker	4 [35, 39, 43, 47]	1.19 (0.85, 1.53)	0.505 (0)	0.77	0.73
Smoker	3 [35, 39, 47]	1.8 (0.54, 5.99)	<0.001 (94.5%)	0.72	1

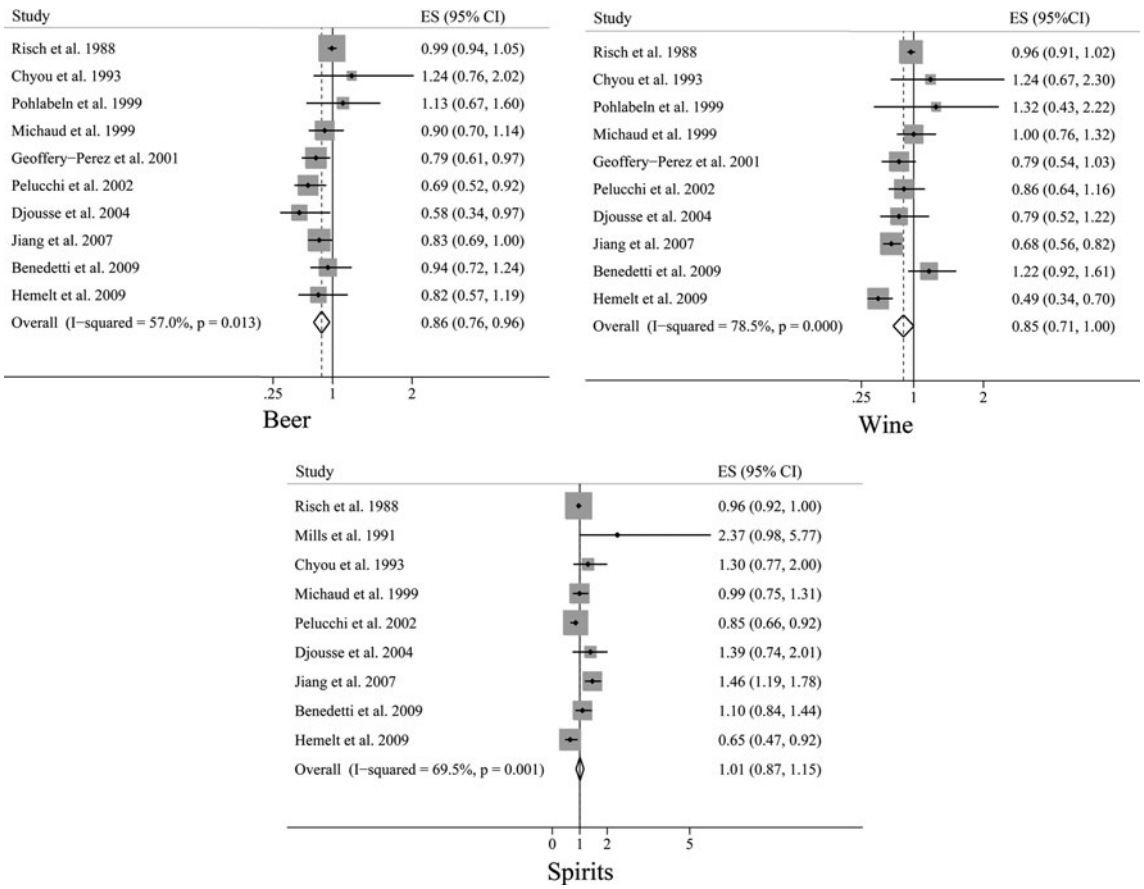

Fig. 2 Forest plots showing the risk estimates of each study and the pooled risk estimates for specific alcoholic beverages

heterogeneous. There was no evidence of significant publication bias either with the Egger's or Begg's test in any subgroup.

Because the consumption of beer and wine was associated with reduced risk of bladder cancer, we further performed the dose–response analysis for these alcoholic beverages. The dose–response meta-analysis included seven studies [41, 43, 45, 47–49, 51] for beer and eight studies [41, 43–45, 47–49, 51] for wine. Figure 3 shows the dose–response relationship between risk of bladder cancer and alcohol consumption from beer and wine. An increase in alcohol consumption of 10 g/day from beer

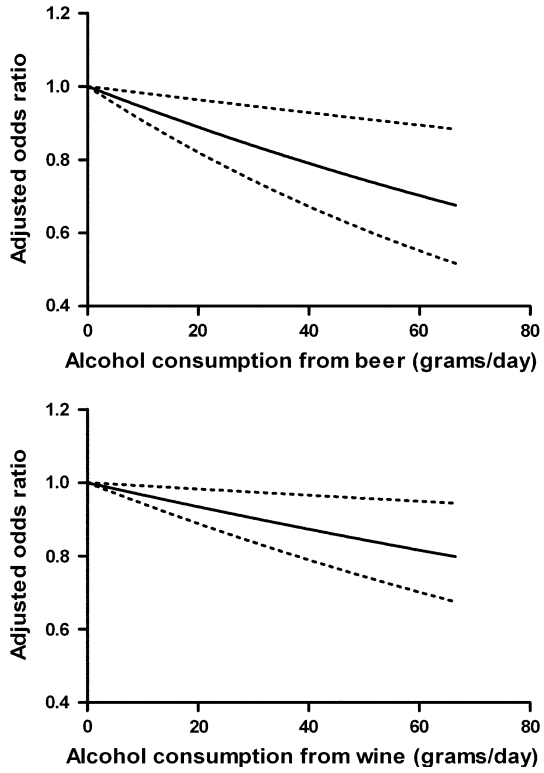

Fig. 3 Odds ratio for bladder cancer by doses of alcohol intake from beer and wine based on the results of the dose–response meta-analyses. *Solid line* represents the estimated odds ratios and the *dotted lines* represent the 95% confidence intervals

(approximately 300 ml per day) was statistically significantly associated with a 5.7% (95% CI 1.8, 9.4%, $P_{\text{heterogenity}} = 0.002$). Similarly a 3.3% decrease risk of bladder cancer for a 10 g/day increase for alcohol consumption from wine (approximately 125 ml per day, 95% CI 0.9, 5.7%, $P_{\text{heterogenity}} = 0.02$) was observed.

In meta-regression analysis, we explored the influence of publication year, geographical region, study design, and method of alcohol assessment in the heterogeneity. However, none of these above was identified as a possible source of heterogeneity among all the included studies.

We also performed sensitivity analyses by sequentially excluding one study in each turn to examine the influence of a single study on the overall estimate or in any strata. The results showed that none of the study could considerably affect the summary of risk estimates in our meta-analysis (data not shown).

Discussion

In this pooled analysis of published cohort and case–control studies, no significant association was observed for alcohol intake overall with bladder cancer. This finding is consistent with the results from the previous meta-analysis by Zeegers et al. [6]. However, our study has several strengths. All studies included in our meta-analysis provided OR estimates controlled for a common set of variables (age, sex and smoking) that are known to be related to bladder cancer, thereby underscoring the independence of association of alcohol consumption with bladder cancer. We included 10 studies published after 1999 that were not included in the previous meta-analysis. A completely novel finding of the present study was that we found support for a negative relationship between beer and wine consumption and the likelihood of bladder cancer, and an increase in alcohol consumption of 10 g ethanol per day from beer (approximately 300 ml per day) or wine (approximately 125 ml per day) was statistically significantly associated with a 5.7 or 3.3% decrease in risk.

The results of the studies included in this analysis were heterogeneous, likely reflecting differences among study populations, model selection, analytic methodology, exposure assessment. We conducted a meta-regression analysis to assess the effect of publication year, geographical region, study design, and alcohol assessment on the heterogeneity. However, none of the confounding factors could explain heterogeneity between the individual studies. Our results from subgroup analysis also could not demonstrate any stratified association. It's been known for a while that smoking is the greatest risk factor for bladder cancer. Although all the risk estimates in this meta-analysis have been adjusted for smoking, how each study controlled for cigarette smoking is different (Table 1). The ORs from older studies may not be adjusted for smoking properly, reflecting that the ORs reported in older studies seems to be higher than those reported in recently published studies, even though publication year was not identified as a source of heterogeneity. Although we cannot reject the possibility that our estimates were distorted because of residual confounding, the overall current literature on alcohol consumption and the risk of bladder cancer suggests no association. Further research is required to elucidate the roles of genetic and environmental factors that may modify the alcohol–bladder cancer association.

The negative associations of bladder cancer with beer and wine, which have lower alcohol content, may attribute to certain ingredients rather than ethanol intake itself. Xanthohumol, the major prenylated flavonoid present in the hops and a common ingredient of beer, has gained considerable interest due to its potential cancer chemopreventive effect [52]. In our previous study, a poly-phenol named Resveratrol, which is abundant in red wine, has been demonstrated to inhibit bladder cancer cells both in vitro and in vivo [53].

As a meta-analysis of previously published observational studies, our study has several limitations that need to

be taken into account when considering its contributions. First, because we did not attempt to uncover unpublished observations and not include studies with insufficient information to estimate an adjusted OR, which could bring publication bias even though no significant evidence of publication bias was observed in Egger's and Begg's test. Second, both volume of alcohol consumption and patterns of drinking have been shown to influence alcohol-related burden of disease, while most of the included studies did not provided data on alcohol intake over time or life drinking patterns. Consequently, we did not have sufficient data to evaluate the risk of bladder cancer associated with these other dimensions of alcohol intake. Third, the limited number of studies and the heterogeneity in the dose–response meta-analysis could possibly result in inaccurate estimates.

In summary, this study applied a detailed meta-analytic approach for combining OR estimates from studies on the relationship between bladder cancer incidence and alcohol consumption. Overall, no association between alcohol consumption and bladder cancer was observed, even stratified by sex, study design, and geographical region, while beer and wine intake could decrease risk for bladder cancer in a dose–response manner. Future research to confirm these findings, and to determine the likely biological mechanism, is warranted.

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