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Relationship between alcohol consumption and the risks of liver cancer, esophageal cancer, and gastric cancer in China

Meta-analysis based on case–control studies

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Abstract

Objective : To study the correlation between alcohol consumption and the risks of liver, esophageal squamous cell carcinoma (ESCC), and gastric cancers in China mainland by meta-analysis.

Methods : We systematically searched electronic databases to identify the case–control studies that reported the association between alcohol consumption and the risks of liver, ESCC, and gastric cancers from January 1, 2010 to April 1, 2020. The Newcastle–Ottawa Scale (NOS) was used to evaluate literature quality, and I^2 analyzes were used to evaluate the heterogeneity.

Results : A total of 2855-related studies were retrieved. After conditional screening, we included 26 case–control studies for meta-analysis. Meta-analysis showed that alcohol consumption was associated with increased risks of liver, ESCC, and gastric cancers (total pooled odds ratio [OR], 1.83; 95% confidence interval [CI], 1.58–2.11; liver cancer OR, 1.83; 95% CI, 1.39–2.40; ESCC OR, 2.00; 95% CI, 1.66–2.40; gastric-cancer OR, 1.54; 95% CI, 1.10–2.15). Subgroup analysis results showed that the pooled ORs of volume of alcohol consumed, years of drinking, age of starting drinking, and drinking status were 1.71 (95% CI, 1.36–2.15), 1.65 (95% CI, 1.33–2.06), 1.38 (95% CI, 0.98–1.94), and 2.00 (95% CI, 1.42-2.81), respectively. Regression analysis showed that geographical region was a source of heterogeneity.

Conclusion : Alcohol consumption increased the risks of liver cancer, ESCC, and gastric cancers in China. Volume of alcohol consumed, years of drinking, age of starting drinking, and drinking status were all significant factors for these risks.

Abbreviations: CI = confidence interval, EAC = esophageal adenocarcinoma, ESCC = esophageal squamous cell carcinoma, HCC = hepatocellular carcinogenesis, NOS = Newcastle–Ottawa Scale, OR = odds ratio, RR = relative risk.

Keywords: alcohol consumption, esophageal squamous cell carcinoma, gastric cancer, liver cancer, meta-analysis

1. Introduction

The Global Burden of Disease Study 2016 (2016) showed that alcohol consumption is one of the main risk factors for cancer death.[1] Liver cancer, stomach cancer, and esophageal cancer have always been the common causes of cancer death in China. According to Global Cancer Observatory (GLOBOCAN) 2012 data, patients in China with these 3 types of cancer accounted for nearly half of such cancer patients worldwide; liver, gastric, and esophageal cancers had standardized mortality rates of 17.1/100000, 17.5/100000, and 12.7/100000,[2] ranking as the third, second, and fourth most common causes of cancer deaths in China, respectively.[3] As these cancers have caused a heavy disease burden, it is necessary to study their risk factors and strengthen prevention. Meta-analysis of the correlation between alcohol consumption and cancer shows that heavy drinking increases the risks of liver (relative risk [RR], 2.07), esophageal (RR, 4.95), and gastric (RR, 1.21) cancers.[4] Previous studies have not adequately studied the relationship between the Chinese population and these 3 cancers. Other studies have mainly focused on the effects of heavy alcohol consumption; instead, few studies focus on volume of alcohol consumed, years of drinking, age of starting drinking, or drinking status, the relationship between drinking and cancer is studied as a relatively simple 1.

Esophageal cancer has 2 predominant histopathological subtypes: esophageal squamous cell carcinoma (ESCC) and esophageal adenocarcinoma (EAC). There are significant molecular differences at both the genomic and epigenomic levels between ESCC and EAC and these 2 cancer types have different sets of driver genes, mutational signatures, and prognostic biomarkers, which are almost mutually exclusive.[5] Among all patients with esophageal cancer, a large proportion of them are

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ESCC, especially in the high-incidence area (i.e., Central and East Asia Asians). A host of studies have shown that there is a strong positive correlation between alcohol drinking and ESCC.[6–9] However, the association between drinking and EAC weakened.[10,11] Therefore, this article only focuses on ESCC.

To comprehensively assess the association between alcohol consumption and the risks of liver, ESCC, and gastric cancers in China, explore the impact of alcohol-related factors (i.e., volume of alcohol consumed, years of drinking, age of starting drinking, or drinking status) on the risk of developing these 3 cancers, and to provide more evidence with which to establish effective prevention methods, we conducted a meta-analysis of case cancer-control studies on alcohol consumption and these 3 cancers in China over the past decade.

2. Materials and methods

2.1. Literature retrieval strategies

This study followed the Standards for Preferred Reporting Items for Systematic Reviews and Meta-Analyses.[12] PICOS scheme was followed for reporting inclusion criteria. We searched PubMed, the China National Knowledge Infrastructure, the Wanfang database, and the China Hospital Knowledge Database using the Medical Subject Headings terms "(drinking OR alcohol) AND (liver cancer) AND (China OR Chinese), (drinking OR alcohol) AND (esophageal cancer) AND (China OR Chinese), (drinking OR alcohol) AND (stomach cancer OR gastric cancer) AND (China OR Chinese), (drinking OR alcohol) AND (gastrointestinal cancer OR digestive tract cancer) AND (China OR Chinese)" to identify relevant published studies in Chinese and English. The searched was performed for articles published from January 1, 2010 to April 1, 2020.

2.2. Literature inclusion criteria and exclusion criteria

Inclusion criteria were as follows:

1. Chinese- or English-language study;
2. research on people in mainland China;
3. Case–control study;
4. clear sample size, with original research data (including odds ratio [OR] value and 95% confidence interval [CI] fully provided;
5. publication year from January 1, 2010 to April 1, 2020.

Exclusion criteria were as follows:

1. duplicate publications or incomplete information;
2. patient population who (a) did not have liver, ESCC, or gastric cancer but instead had other cancers or non-cancer diseases such as hypertension or diabetes, and (b) were non-mainland Chinese;
3. non-case–control study and/or year of publication outside the match parameters; and
4. OR value and 95% CI not given.

2.3. Literature quality evaluation

The quality of the literature was evaluated according to the Newcastle–Ottawa Scale (NOS). The NOS includes items on population selection (4 items, 4 points), comparability between groups (1 item, 2 points), and exposure evaluation (3 items, 3 points), for a total of 9 points. Scores of 7 to 9 points indicate high-quality studies, those of 4 to 6 points indicate moderate-quality studies, and those of 1 to 3 points indicate low-quality studies. All studies included in this meta-analysis scored greater than 7 points. Any discrepancies between authors we turned to the original literature and relevant experts.

2.4. Information extraction

We screened studies according to the inclusion and exclusion criteria, evaluated literature quality, and then extracted the data. Extracted content included author, year of publication, age of study subjects, type of cancer, case–control matching, number of cases, number of control groups, geographical region, comprehensive statistical-index OR value, and the OR value's 95% CI.

To avoid the omission and duplication of the literature included in this study, we were done separately by 2 people in the process of literature search, screening and information extraction. In addition, to control confounding factors, the effect value OR and 95% CI included in the meta-analysis were adjusted by multivariate or stratified analysis in the original articles.

2.5. Ethics

This study did not involve human beings or experimental subjects. No ethical approval is required.

2.6. Statistical analysis

We used Stata software version 15 (StataCorp, College Station, Texas, US) to perform meta-analysis. Heterogeneity among studies was assessed using I^2 statistics, with $I^2 > 50\%$ representing significant heterogeneity. When the heterogeneity I^2 was $<50\%$ ($P \geq .05$), we selected the fixed-effects model; when the heterogeneity I^2 was $\geq 50\%$ ($P < .05$), we selected the random-effects model. Count data were calculated by pooled OR and 95% CI. We performed subgroup analysis to evaluate the effects of variables relevant to alcohol consumption. To find the source of heterogeneity, we further performed meta-regression with covariables, such as NOS score, matching ratio, and geographical region. Egger test was used to quantitatively assess publication bias. $P < .05$ was considered statistically significant.

This study conducted subgroup analysis on volume of alcohol consumed, years of drinking, age of starting drinking, and drinking status. Grouping was as follows:

1. units of drinking volume were converted to g/d: ≤ 40, 40–79, 80–120, or >120; (2) years of drinking (years) ≤ 30 or >30;
2. age of starting drinking (years) was ≤ 30 or >30; and
3. drinking status was past drinking or current drinking.

3. Results

3.1. Results of literature retrieval and screening

According to the above inclusion and exclusion criteria, we retrieved a total of 2855 articles. After conditional screening, we included 26 articles.[13–38] for meta-analysis, including 11 articles on liver cancer, 10 on ESCC, and 5 on gastric cancer. The literature screening process is shown in Figure 1. The total number of cases was 1643642 cases, with 87309 cases in the case group and 1556333 cases in the control group. All of the participants were age >35 years (Table 1).

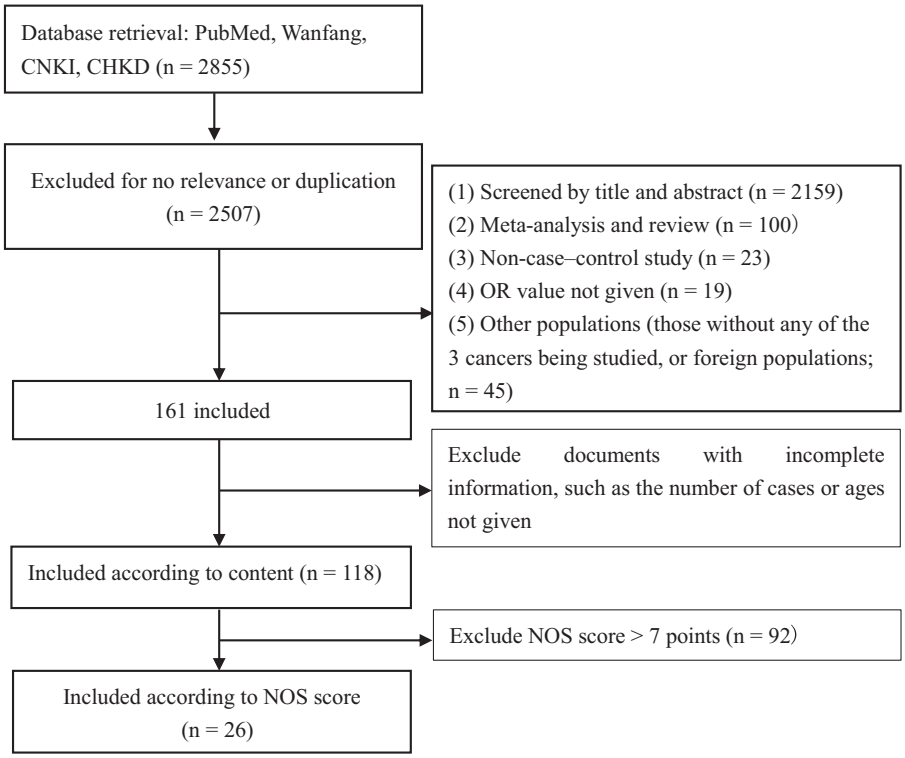

Figure 1. Flowchart of the systematic search of literature on alcohol consumption and the risks of liver, ESCC, and gastric cancers in China.

Table 1

Case–control studies on the association between alcohol consumption and the risks of liver, ESCC, and gastric cancers in China.

Author	Publication year	Type of cancer	Matching	Cases (M_1/N_1)[1]	Controls (M_2/N_2)[2]	Region	NOS score
Long Ji	2010	Liver	Other	394/500	374/507	Guangxi	7
Yujian Lan	2012	Liver	1:1	162/200	162/200	Guangxi	7
Xiaoli Wang	2012	Liver	1:1	215/251	215/251	Guangdong	7
Xiangui Tong	2013	ESCC	Other	133/164	266/328	Anhui	7
Shuping Nie	2012	ESCC	Other	426/612	547/770	Jiangsu	8
Dandan Chen	2013	Liver	Other	237/323	256/443	Henan	7
Haifeng Yu	2014	Liver	1:1	90/104	90/104	Guangdong	7
Yuefen Zhou	2018	Gastric	1:1	148/210	148/210	Zhejiang	8
Ming Wu	2011	ESCC	Other	1191/1520	2916/3879	Jiangsu	7
Xiaorong Yang	2017	ESCC	1.3:1	921/1353	432/1961	Jiangsu	7
Huabin Wu	2012	Liver	1:1	956/1254	956/1254	Jiangsu	9
Qing Zhu	2019	Gastric	Other	166/215	330/430	Gansu	7
Zhongpei Xie	2013	ESCC	Other	114/196	127/201	Guiyue	7
Sa Tang	2014	ESCC	Other	3030/3759	3447/5196	Henan	7
Xueke Zhao	2014	Liver	Other	602/762	458/798	Guizhou	7
Shasha Chen	2013	Liver	Other	98/120	156/199	Guizhou	7
Jianxue Duan	2018	Liver	Other	268/330	353/464	Chongqing	8
Yan Li	2016	Gastric	1: 1	36/71	36/71	Jiangsu	7
Jianli Hu	2010	ESCC	1: 1.5-2	283/283	538/538	Beijing	7
Fansong Meng	2019	ESCC	Other	203/278	406/556	Shandong	7
Ying Liu	2014	Liver	1: 1	812/1007	488/1007	Hebei	7
Hong Lu	2012	ESCC	Other	310/400	565/752	Gansu	7
Qingping Xue	2015	Gastric	1: 1	224/308	224/308	Sichuan	7
Shaoyi Lin	2011	Liver	1: 1	303/388	303/388	Fujian	7
Jia Wang	2012	Gastric	1: 1	347/476	347/476	Jiangsu	7
Weihong Gan	2011	ESCC	1: 2	81/97	162/194	Shanghai	7

1: M_1—Number of male in the case group; N_1—Total number of cases; 2: M_2—Number of male in the control group; N_2—Total number of controls.

3.2. Meta-analysis of the literature on the relationship between alcohol consumption and the risks of liver, esophageal squamous cell carcinoma, and gastric cancers

Meta-analysis results showed that alcohol consumption was associated with increased risks of liver, ESCC, and gastric cancers (total pooled OR, 1.83 [95% CI, 1.58–2.11]; liver cancer OR, 1.83 [95% CI, 1.39–2.40]; ESCC OR, 2.00 [95% CI, 1.66–2.40]; gastric-cancer OR, 1.54 [95% CI, 1.10–2.15]). The difference between drinking and the 3 cancers types was statistically significant ($P < .05$). A forest plot of the relationship between alcohol consumption and liver, ESCC, and gastric cancers is shown in Figure 2.

3.3. Subgroup analysis of alcohol-related variables and liver, esophageal squamous cell carcinoma, and gastric cancers

We performed a subgroup analysis based on volume of alcohol consumed, years of drinking, age of starting drinking, and drinking status. The combined-effect OR values were 1.71 (95% CI, 1.36–2.15), 1.65 (95% CI, 1.33–2.06), 1.38 (95% CI, 0.98 – 1.94), and 2.00 (95% CI, 1.42–2.81), respectively. The greater the daily alcohol consumption, the greater the OR value of cancer risk. Drinking <40 g daily had no statistical significance to the risk of cancer (OR, 1.50 [95% CI, 0.94–2.40]; $I^2 = 93.7\%$, $P > .05$). Risk was greatest when daily volume of alcohol consumed exceeded 120 g / d (OR, 2.44 [95% CI, 1.96–3.02]; $I^2 = 85.0\%$, $P \leq .001$). Drinking for >30 years, beginning to drink at >30 years old, and past drinking were all statistically significant (Table 2).

3.4. Meta-regression analysis of non-research variables (Newcastle–Ottawa Scale score, matching, and geographical region)

The heterogeneity test showed that $I^2 = 82.2\%$ and $P \leq .001$. This large degree of heterogeneity suggested the data were original. To

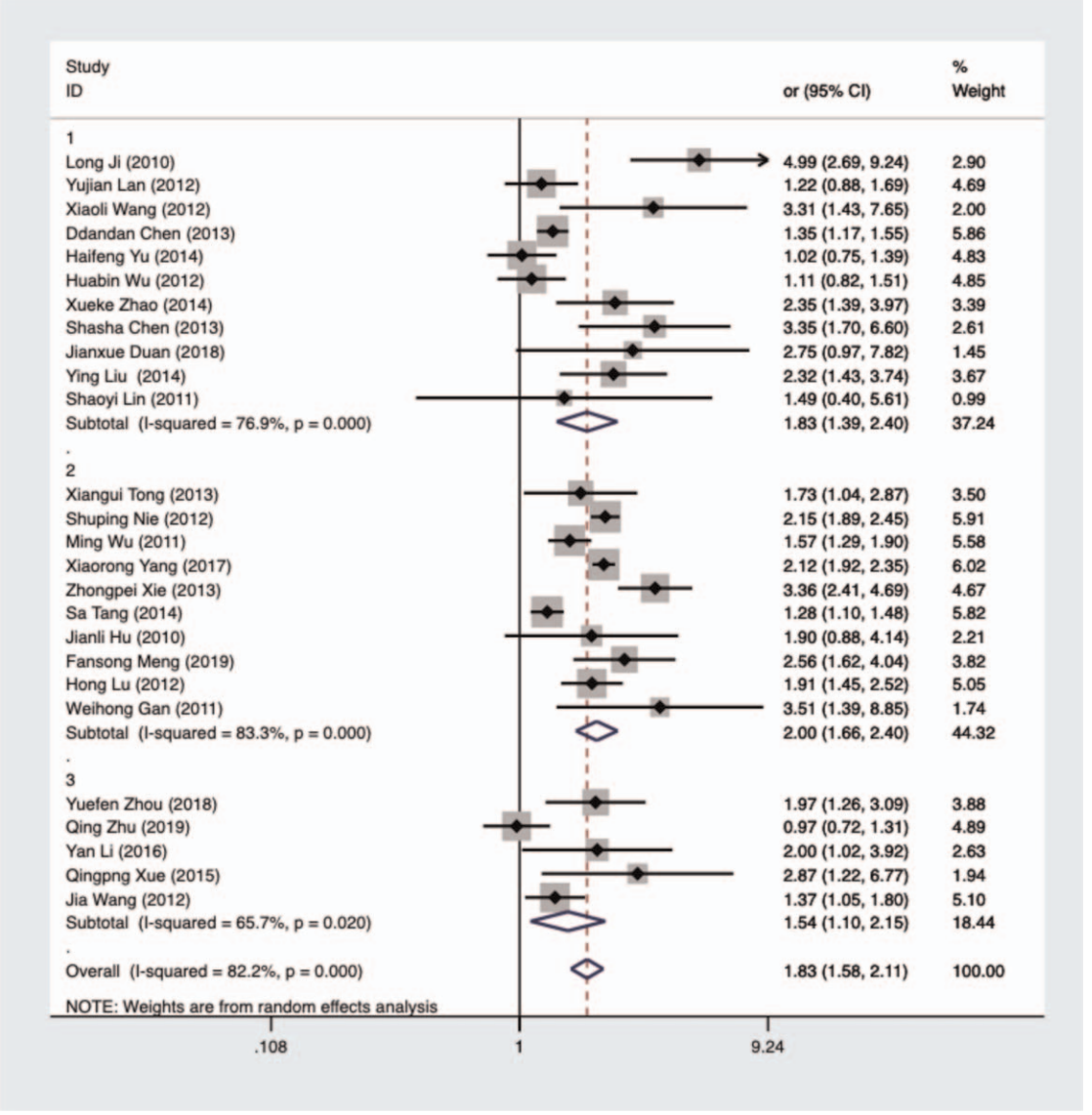

Figure 2. Forest plot of analyzed literature on the relationship between alcohol consumption and liver, ESCC, and gastric cancers in China. 1—liver cancer; 2—ESCC; 3—gastric cancer.

Table 2

Subgroup analysis of meta-analysis between alcohol consumption and the risks of liver, ESCC, and gastric cancers.

Subgroup	Cases	Controls	OR	95% CI	I^2 (%)	P
Volume of alcohol consumption (g/d)						
≤40	4176	6679	1.50	(0.94–2.40)	93.7	.091
40–79	449	845	1.58	(1.09–2.28)	78.7	.016
80–120	533	804	1.96	(1.44–2.68)	00.1	.000
≥120	686	854	2.44	(1.96–3.02)	85.0	.000
Total	5844	9182	1.71	(1.36–2.15)	90.4	.000
Years of drinking						
≤30	1502	2682	1.26	(0.99–1.60)	68.0	.056
>30	1663	2281	2.31	(1.60–3.34)	90.8	.000
Total	3165	4963	1.65	(1.33–2.06)	85.5	.000
Age of starting drinking						
≤30 yrs old	1340	2221	1.28	(0.79–2.08)	97.2	.315
>30 yrs old	508	691	1.55	(1.02–2.36)	91.0	.040
Total	1848	2912	1.38	(0.98–1.94)	96.0	.061
Drinking status						
Past drinking	1151	2378	2.24	(1.35–3.70)	76.0	.006
Current drinking	1574	3164	1.84	(1.00–3.40)	94.4	.000
Total	2725	5542	2.00	(1.42–2.81)	89.8	.000

Table 3

Meta-regression analysis of NOS score, matching ratio, and geographical region.

Variables	OR	95%CI	P	P_a
NOS score	1.06	(0.86–1.25)	.66	0.95
Matching	1.04	(0.92–1.02)	.18	0.43
Regions	1.03	(1.01–1.04)	.03	0.04

P_a: adjusted *P* value.

determine the difference between drinking and the risks of these 3 cancers, we conducted a meta-regression analysis to explore the combined-effect size of non-study variables (NOS score, matching ratio, and geographical region) on our meta-analysis of drinking and liver, ESCC, and gastric cancers. The NOS scores included in the regression analysis were all ≥7 points. Matching ratio groups were 1: 1, 1: 2, 1: 1.5–1.2, 1.3: 1, and other. The regression model showed a good fit ($\tau 2 = 0.098$), and the heterogeneous performance accounted for 91.10% of the residual variance. The meta-regression results suggested that geographical region was a source of heterogeneity ($P = .03 < .05$) (Table 3).

3.5. Publication bias

The results of Egger test indicated that there was no significant publication bias observed in the selected studies ($P = .518$) (Fig. 3).

4. Discussion

This meta-analysis included 26 case–control studies on alcohol consumption and the risks of liver, ESCC, and gastric cancers in China. The results showed that drinking alcohol was a risk factor for all 3 types of cancer (pooled OR, 1.83 [95% CI, 1.58–2.11]). The forest plot in Figure 2 shows that drinking increased the risk of and was significantly related to ESCC (OR, 2.00 [95% CI, 1.66–2.40]), liver cancer (OR, 1.83 [95% CI, 1.39–2.40], and gastric cancer (OR, 1.54 [95% CI, 1.10 –2.15]). Another study found that among all types of cancer caused by alcohol consumption, liver cancer has the highest mortality rate and disease burden ratio.[39] Evidence for the relationship between alcohol consumption and gastric cancer is not clear, but excessive drinking is currently considered one of the risk factors for this cancer.[40] The carcinogenic mechanism of alcohol consumption is currently not fully understood. Acetaldehyde is the first metabolic product of ethanol, which modifies DNA by producing DNA adducts and induces oxidative stress and genetic changes in the function of alcohol-metabolizing enzymes, thereby exerting carcinogenic and mutagenic effects. It is part of the carcinogenic effect of drinking on the liver and upper digestive tract.[41]

At present, it is believed that alcohol causes hepatocellular carcinogenesis (HCC) mainly through 3 aspects. On the one hand, alcohol consumption as an inducer and promoter of liver cancer to promote the occurrence of it.[42] Long-term alcohol intake can reduce the liver's detoxification function, reduce the intake of nutrients and reduce the body's immunity. Secondly, acetaldehyde, the intermediate metabolite of alcohol, is considered to be the positive factor for the occurrence of HCC. Modern genetic studies have found that acetaldehyde induces HCC as the target of acetaldehyde dehydrogenase 2 gene (ALDH2) on chromosome 12.[43] Thirdly, heavy alcohol consumption may cause alcoholic cirrhosis, which may further develop into HCC.[44,45] Study has found that the human liver can only completely metabolize 80 mL of ethanol per day, and the consumption of more than 80 mL will increase and accumulate

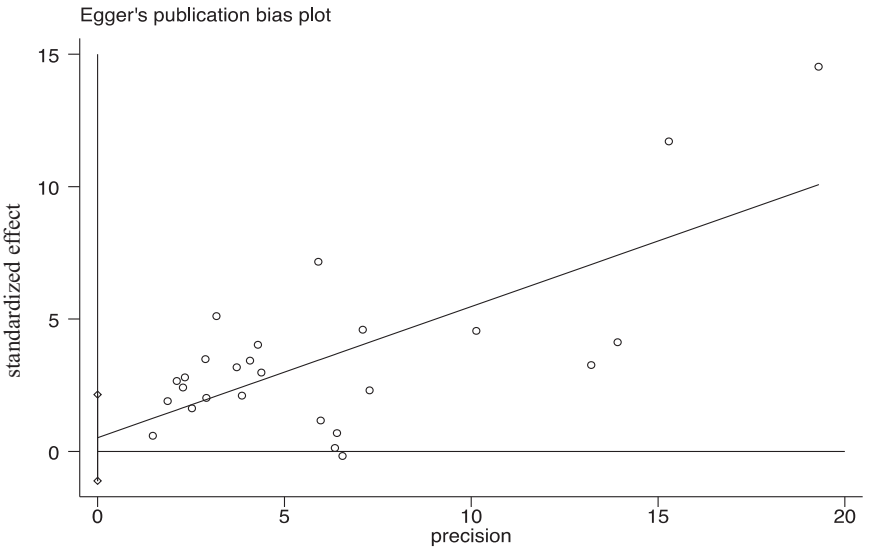

Figure 3. Results of Egger test for publication bias.

the concentration of acetaldehyde in the blood. Acetaldehyde has the role of carcinogenicity and gene mutation and plays a major role in the process of alcohol-related carcinogenesis.[46]

This study found a positive dose relationship between alcohol consumption and liver, ESCC, and gastric cancers. As volume of alcohol consumed daily increased, so did the risk of cancer, which was similar to the results of similar studies. There is a certain dose relationship between alcohol consumption and the incidence of esophageal cancer.[47] Alcohol intake increases the risk of esophageal cancer according to the daily ethanol intake, the type of alcoholic beverages consumed, time of abstinence, age of starting drinking, population, and differences in esophageal-cancer subtypes.[48] The relationship between alcohol consumption and cancer is still controversial, especially when consumption is light or moderate.[49] Our results showed that drinking <40 g daily was not statistically significant to the risk of cancer; the risk was greatest when volume of alcohol consumed daily was >120 g / d. This differed somewhat from the results of similar studies.[50,51] In addition, a meta-analysis of the relationship between alcohol consumption and cancer that included 222 studies found that in studies conducted only in Asian populations, the effect of low alcohol consumption on esophageal-cancer risk was statistically significant (RR, 1.49 [95% CI, 1.12–1.98]).[52] This suggests to some extent that Asians have more genetic polymorphisms encoding alcohol-metabolizing enzymes than other populations do, and therefore the amount of alcohol consumed in these populations should be controlled within an appropriate range.

Our study found that subjects who had been drinking for more than 30 years had a cancer risk OR value greater than those who had been drinking for less than 30 years, which might be related to long-term drinking and increased alcohol consumption. Studies have found that the risk of developing esophageal cancer increases along with alcohol consumption.[53] Compared with not drinking throughout life, the risk of esophageal cancer is positively correlated with the amount of alcohol consumed, and moderate and high levels of drinking are associated with an increased risk of esophageal cancer.[54] A summary analysis of 7 studies (5 case–controls and 2 cohort studies) conducted by the International Barrett and Esophageal Adenocarcinoma Federation (BEACON) found that an increase in years of drinking was associated with an increased risk of esophageal cancer.[55] Another study found that drinking frequency was positively correlated with the risks of esophageal and liver cancers. There is a positive dose–response relationship between amount of alcohol consumed and the risks of esophageal and liver cancers. People who drink ≥50 g/day on average have an increased risk of stomach cancer.[56] Our study found that the combined effects of the 3 types of cancer with an age of starting drinking ≥30 years was greater than when the age of starting drinking was <30 years. The effect of the age at which a person starts drinking on the risk of ESCC is uncertain. Studies have found that as they increase their daily alcohol intake, people who start drinking at an old age are more likely to suffer from ESCC.[57] In Castellsague et al study,[58] people who started drinking alcohol at an older age were more likely to develop esophageal cancer as their daily alcohol intake increased. However, there are also studies showing no association between age of beginning drinking and the risk of developing ESCC.[59]

This study could only search in Chinese and English databases, and no literature in other languages was collected, which may be influenced by the bias of language selection. Most of the results of heterogeneity tests indicated that there was significant heterogeneity among the studies, which might be related to the design of the studies, the variety and number of confounding factors controlled, and ethnic or regional differences. We conducted a further meta-regression analysis, the results of which showed that geographical region was the source of heterogeneity. The source areas of the literature included in this article were Shanxi, Guangxi, Guangdong, Anhui, Jiangsu, Henan, Zhejiang, Gansu, Guizhou, Chongqing, Beijing, Shandong, Sichuan, Fujian, and Shanghai, in China. Incidences and mortality rates of liver, ESCC, and gastric cancers vary from region to region,[60] which might explain the high degree of heterogeneity among the studies we analyzed. Another source of heterogeneity may be the exclusion of the earlier literature on this topic. The methods of diagnosis and treatment of cancer are changing rapidly, and the diagnostic criteria reported in the earlier literature are somewhat different

from those reported in the more recent literature, as are the detection rates and survival rates. For example, simple esophagectomy is associated with a higher recurrence rate, with a low 5-year survival rate of 5 to 34 percent.[61] Recent advances in the treatment of patients requiring esophagectomy have been neoadjuvant chemoradiotherapy or chemotherapy. Randomized controlled trials have shown that neoadjuvant chemoradiotherapy or chemotherapy provides a survival benefit for both types of esophageal cancer compared to surgery alone.[62–64]

Besides, the occurrence of cancer is the result of many factors. Drinking alcohol is a known and major cause of esophageal squamous cell carcinoma, especially in countries with low incidence. However, in high-incidence areas of Asia and Africa, other risk factors may be more important, including poor diet, indoor air pollution, consumption of hot drinks, poor oral health, use of non-tap water and use of opium.[65] A case–control study in the Islamic Republic of Iran, which measured exposure to PAHs in endoscopically normal esophageal tissues from cases of oesophageal squamous cell carcinoma and controls, reported odds ratios of more than 25 for the most exposed quintile compared with the least exposed quintile.[66] According to the previous studies the important risk factors for gastric cancers are the host's genetic makeup, the characteristics of H. pylori strains, and environmental factors, notably diet.[65] Globally, hepatitis B virus infection was responsible for 33% of deaths from liver cancer, alcohol consumption for 30%, hepatitis C virus infection for 21%, and other causes for 16%, with significant variation in the underlying etiologies among regions and countries.[67]

This study showed that the combined effect of drinking in the past was greater than that of current alcohol consumption. This was consistent with the Gao Shan[68] study, which found that drinking is associated with an increased risk of liver cancer, but other studies have shown that current alcohol consumption poses a greater risk of cancer.[69] Long-term alcohol intake can reduce the liver's detoxification function and the body's nutritional intake and immunity, and then induce and accelerate liver cancer risk factors to promote the occurrence of liver cancer.[70] This suggests that the effects of drinking, whether past or current, on cancer cannot be ignored.

The strengths of this study lay in the analysis of drinking and its more related subgroup factors. To date, this study provides complete and recent evidence on the association between alcohol drinking and liver, ESCC, and gastric cancers. We were able to explore the relationship between drinking and the risks of these 3 cancers across many aspects, not limited to study on alcohol consumption. Our study results can provide a specific basis for cancer prevention methods. In addition, this analysis focused on important disease burden factors such as digestive-tract tumors and drinking in China, which has practical value for providing new ideas for the study of key tumors and their risk factors.

The limitations also exist. The occurrence and development of liver cancer, ESCC and gastric cancer are the result of a combination of factors, such as genes, smoking, dietary habits, and exposure environment. We included only case–control studies of alcohol consumption and the supporting evidence was weak. More rigorous scientific research such as prospective cohort studies are needed to continue to analyze the relationship between alcohol consumption and cancer risks. Second, men have more opportunities to drink alcohol than women, but this paper did not compare men and women alcohol consumption due to the lack of data from gender analysis in the original articles.

In conclusion, alcohol consumption increased the risks of liver cancer, ESCC, and gastric cancer in China. Volume of alcohol consumed, and years of drinking were related to all 3 types of cancer. The risk of cancer increased along with daily alcohol intake. Our results suggested that controlling alcohol consumption and reducing the years of drinking could help prevent the occurrence of liver, ESCC, and gastric cancers, and that in particular people age >30 years and populations with a high incidence of cancer should reduce their alcohol consumption.

Author contributions

Conceptualization: Baohua Wang.
Methodology: Yuting Sha.
Writing – original draft: Fengdie He.

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