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BRIEF REPORT

Moderate alcohol drinking might be protective for systemic lupus erythematosus: a systematic review and meta-analysis

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Abstract Conflicting evidence for the effect of moderate alcohol drinking on the development of systemic lupus erythematosus (SLE) existed at present. In the current study, we performed an extensive search of relevant studies and performed a meta-analysis to obtain a more precise estimate. Thirty-eight studies were identified from electronic databases and chosen for detailed review, then six articles from six case–control studies with one cohort study were included in our meta-analyses. Meta-analyses were divided into two subgroups in which patients in the study of Washio et al. treated for less than 5 years (subgroup A) or less than 10 years (subgroup B) were involved, respectively. The odds ratio (OR) of moderate alcohol drinking in the meta-analyses of subgroup B for the development of SLE was significantly decreased (OR 0.723, 95% confidence interval (95% CI) 0.547–0.954), while moderate alcohol drinking in the meta-analysis of subgroup A did not demonstrate a decreased risk of SLE (OR 0.780, 95% CI 0.491–1.240). Meta-analyses of six case–control studies in the two subgroups both demonstrated that moderate alcohol drinking had a protective effect on the development of SLE. Taken together, our results show that moderate alcohol drinking might be protective for SLE.

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Introduction

Systemic lupus erythematosus (SLE) is an autoimmune disease characterized by typical involvement of many different organ systems and immunological abnormalities [1]. The precise etiology of SLE remains unclear. Over the past few decades, the familial occurrence of SLE has been increasingly recognized, which indicated that genetic susceptibility plays an important role in the etiology of SLE. However, with heritability of SLE estimated to be around 60%, environmental factors must also play a role in the pathogenesis of SLE [2]. Up to now, many environmental factors have been suspected of inducing SLE, but the role of these nongenetic stimuli has remained poorly understood. Presently, commonly accepted environmental etiological factors regarding SLE include ultraviolet light and drugs [3]. Others, including tobacco smoking, alcohol drinking, hair-coloring products, silica and dietary factors, might be associated with SLE [4].

Among these environmental factors, studies regarding the effects of tobacco smoking or alcohol drinking on the development of SLE had shown conflicting results [4]. According to the relationship of smoking and the development of SLE, a meta-analysis had been undertaken and obtained a significant association between current smoking and the development of SLE, but no association between past smoking and the development of SLE was found [4]. Additionally, tobacco smoking and alcohol drinking are known to be correlated, and alcohol consumption was shown to be inversely associated with the risk of SLE in

several studies [5, 6]. Nevertheless, the relationship of alcohol drinking with the risk of SLE remains controversial.

Meta-analysis is a powerful tool for summarizing the results from different studies by producing a single estimate of the major effect with enhanced precision. One of the major advantages of meta-analysis is to increase sample size, which may reduce the probability that random error will produce false-positive or false-negative associations [7].

In the present study, to better quantify the magnitude of the association of alcohol drinking with the development of SLE, we undertook a meta-analysis on it.

Materials and methods

Identification of studies

Our search sought published studies for all years available, mainly in PubMed, Medline database, and the Cochrane Collaboration database using the following keyword combinations: "alcohol" and "systemic lupus erythematosus"; "environmental" and "systemic lupus erythematosus"; "alcohol" and "autoimmune diseases"; "risk factor"; and "systemic lupus erythematosus". We also search the dissertation and conference papers. References in the studies were reviewed to identify additional reports. Moreover, we contacted experts in the field to identify any unpublished related studies. At the same time, since alcohol drinking and cigarette smoking were often investigated in the same study, we searched some studies about the relationship between smoking and SLE; then, we contacted those researchers to get to know if they had investigated alcohol drinking. If they had, we attempted to obtain related information. We also performed hand searches of bibliographies of some key articles on SLE of full text in the library.

Inclusion and exclusion criteria

Inclusion criteria were as follows: (1) case–control or cohort study; (2) alcohol drinking was examined as a related factor for the development of SLE in humans; and (3) the measure of effect with its variance was provided or could be calculated indirectly.

Exclusion criteria were as follows: (1) animals study; (2) case report, review, descriptive, or qualitative research; (3) fewer than five participants were involved in the study; (4) the effect of alcohol drinking was not investigated; and (5) alcohol drinking was examined as a prognostic factor.

Data extraction

Studies were appraised for quality using the Critical Appraisal Skills program including sample size, participant characteristics at baseline and on completion, attrition rates and reporting and the application of controls or multiple measures. The following information were extracted from each study: (1) first author and year of publication; (2) study design; (3) sample size; (4) populations from which patients and controls were selected; (5) definition of alcohol drinking and diagnosis of SLE; (6) frequencies of alcohol drinking in both case and control groups; (7) adjusted odds ratio (OR) or relative risk (RR) of SLE developing with 95% confidence interval (95% CI); and (8) potential confounders controlled for.

Statistical analysis

Statistical manipulations were conducted using Stata statistical software, release 9.0 (College Station, Texas: Stata Corporation, 2005).

Heterogeneity between studies was assessed by testing Cochran's Q statistic. This heterogeneity test assessed the null hypothesis that all studies were evaluating the same effect. P values less than 0.05 indicated heterogeneity across studies. When the test found significant study heterogeneity, the random effects model was used for meta-analysis and assumed that the studies were a random sample of a hypothetical population of studies. The pooled estimate of OR was obtained by the Dersimonian–Laird method in the random effect model. Pooled OR in the meta-analysis was performed weighting individual OR with the inverse of its variance.

A funnel plot was generated by plotting the OR of SLE developing (X-axis) against the standard error in each study (Y-axis) to explore the potential for publication bias. If the plot was symmetrical, publication bias did not exist. The symmetrical funnel plot was tested by Egger's test, which is a linear regression approach. The standard normal deviate (SND), defined as the odds ratio divided by its standard error, is regressed against the estimate's precision, the latter being defined as the inverse of the standard error (regression equation: $\mathrm{SND}=a+b\times\mathrm{precision}$) [8]. If the line ran through the origin at standard normal deviate zero ($a=0$) without significance ($P>0.1$), it would suggest that the funnel plot was symmetrical.

A sensitivity analysis was performed by omitting each study at each time from the meta-analysis to illustrate the accuracy and stability of the analytic results.

Results

After we contacted the authors of some studies about the association between cigarette smoking and SLE, we knew that alcohol drinking was not investigated in a standardized manner in several studies. One study had investigated it without significance, but the researcher did not give us the raw

data from which the OR and 95% CI could be calculated. No reply was obtained from researchers of the other studies. Of the 3,455 studies identified by our literature search, 38 were chosen for detailed review. Based on the inclusion and exclusion criteria, six articles including six case–control studies and one cohort study (in which one article included two case–control studies) were included in the meta-analysis (Table 1).

Study designs and demographics

Study populations resided in four different countries, where three studies were from Japan [6, 9], two from America [10, 11], one from England [5], and one from Sweden [12]. Although the SLE patients of five studies [6, 9, 10, 12] were female and those in the other two studies [5, 11] were not restricted to female, the controls in these two studies [5, 11] were all matched to SLE cases for sex.

SLE diagnosis in all the studies was based on the classification criteria established by the American College of Rheumatology [13, 14]. The cases of four studies [6, 9, 11] among six case–control studies were outpatients, while the cases of the other two studies [5, 12] were a little different from them. In the study of Bengtsson et al. [12], the SLE patients were retrieved by the use of computerized

Table 1 List of the original information of seven studies included in meta-analysis

Author (reference)	Location/population	No. of cases/controls	Population of controls	No. of alcohol drinking of cases/controls	OR (IRR) and 95% CI
Case–control studies					
Washio et al. [9]	Kyushu of Southern Japan, women only	78/329	Nursing college students and care workers in nursing homes	Median duration	Median duration
				<5 years	<5 years
				<1 day/week: 22/284	1.00
				1–3 days/week: 8/37	2.33 (0.94–5.82)
				>3 days/week: 7/6	8.22 (2.21–30.50)
				<10 years	<10 years
				<1 day/week: 57/284	1.00
				1–3 days/week: 11/37	1.29 (0.60–2.77)
				>3 days/week: 9/6	4.49 (1.43–14.08)
	Hokkaido of Northern Japan, women only	35/188	Participants of a health checkup in local area	Median duration	Median duration
				<5 years	<5 years
				<1 day/week: 15/144	1.00
				1–3 days/week: 3/24	0.60 (0.14–2.60)
				>3 days/week: 6/18	1.20 (0.33–4.31)
				<10 years	<10 years
				<1 day/week: 22/144	1.00
				1–3 days/week: 4/24	0.60 (0.16–2.22)
				>3 days/week: 9/18	1.34 (0.44–4.34)
Bengtsson et al. [12]	Southern Sweden, women only	85/205	General population in the area	0 g/month: 33/54	1.00
				≤150 g/month: 32/73	0.70 (0.30–1.30)
				>150 g/month: 20/78	0.40 (0.20–0.80)
Ghaussy et al. [11]	US, New Mexico	125/125	General medical outpatients	0 drinks/week: 93/105	0.7, $P>0.5$; 95% CI were not reported
				1–2 drinks/week: 25/11	
				3–5 drinks/week: 5/6	
				>6 drinks/week: 2/3	
Hardy et al. [5]	Nottingham of UK	150/300	General population registers in the area	0 unit: 63/75	1.00
				1–2 units: 28/50	0.73 (0.39–1.36)
				3–5 units: 19/57	0.41 (0.20–0.85)
				6–10 units: 25/60	0.47 (0.24–0.91)
				>10 units: 14/56	0.30 (0.14–0.63)
Nagata et al. [6]	Japan, women only	282/292	Females with appointments at same public health center	Never: 257/254	1.00
				Weekly: 12/26	0.52 (0.25–1.06)
				Daily: 5/10	0.57 (0.19–1.71)
Cohort study					
Formica et al. [10]	US, African-American, women only	67/53,924	53,924 African-American women	0 drinks/week: 36[a]	1.00
				<1 drink/week: 2[a]	0.50 (0.10–3.60)
				1–6 drinks/week: 16[a]	1.30 (0.70–2.40)
				>6 drinks/week: 3[a]	0.80 (0.20–2.60)

[a] The frequency of alcohol drinking in the control group was not reported.

diagnosis registers where outpatients and inpatients were registered. In the study of Hardy et al. [5], the majority of SLE patients were outpatients and the remaining were from the same area presenting at least 2 years before the investigation.

Controls were the general population of the same area in only one case–control study [12] and were the participants of a health examination in three case–control studies [5, 6, 9]. None of the case–control studies used inpatient hospital-based controls.

The definitions of alcohol drinking in different studies were not the same unfortunately. In two of seven studies, alcohol drinking was defined by the frequency of day per week and those who drank more than 1 day/week were defined as having a drinking habit [9]. In five studies [5, 6, 10–12], alcohol consumption was measured using the unit of intake, but the units of measurement were different across countries. Alcohol consumption was measured either by units or by frequency in the week preceding the interview in the study of Hardy et al. [5], and alcohol drinking was defined by the total number of drinks per week in the past year by using the midpoint of each category in the study of Formica et al. [10]. Alcohol consumption was categorized by the intake frequency per week into never, weekly, and daily in the study of Nagata et al. [6].

Statistical analyses of all studies were limited to alcohol drinking that preceded the diagnosis of SLE. Only two studies [9] reported the median duration that was from the time when SLE was diagnosed to the study time and performed two types of analyses which included patients treated for less than 5 years and patients treated for less than 10 years. Although the alcohol consumption was divided into several ranks in all studies, a test for the trend of alcohol consumption provided an evidence of a dose–response relationship in only two studies [5, 6].

In five studies [5, 9, 11, 25], the statistical method was same with multivariate conditional logistic regression. Multivariate unconditional logistic regression and Cox proportional hazards regression were used in the other two studies of Nagata et al. [6] and Formica et al. [10], respectively. The confounders controlled for in the analyses were different. The analyses were all adjusted for smoking combined with age [9, 11], body mass index [12], or other demographic variables [6, 10, 11]. Social class was adjusted in the study by Hardy et al. [5].

Results of the meta-analysis

In order to lessen the variance between those studies, we redefined the moderate alcohol drinking coordinately among them based on criteria [15, 16]. Moderate alcohol drinking was redefined in those studies respectively as follows: from 1 to 3 days/week of alcohol drinking in the study of Washio et al. [9], from one to six drinks/week in the study of Formica et al. [10], more than 0 and less than 150 g/month in the study of Bengtsson et al. [12], from three to five drinks/week in the study of Ghaussy et al. [11], from 3 to 5 units in the week preceding interview in the study of Hardy et al. [5], and weekly alcohol drinking in the study of Nagata et al. [6].

Additionally, two types of patients with different median durations (less than 5 years and less than 10 years) were analyzed, respectively, in the two studies of Washio et al. [9]. We performed two types of meta-analyses in which the results of patients treated for less than 5 years (subgroup A) and less than 10 years (subgroup B) were included, respectively.

The odds ratios with their 95% CI of moderate alcohol drinking with SLE in subgroup A and subgroup B were listed in Table 2.

Meta-analysis of subgroup A

For the odds of SLE in moderate alcohol drinking vs never or nearly never alcohol drinking, the pooled OR estimate for these seven studies was 0.780 (95% CI was 0.491–1.240, $P>0.05$) using the random effects model. Heterogeneity among them was tested with significance ($Q=14.039$, $P=0.029<0.05$). The funnel plot was Fig. 1. The Egger's test suggested that the publication bias did not exist (the value of a was 0.935, $P=0.293>0.05$). The sensitivity analysis found that the OR of the study of Washio et al. [9] had the greatest influence on the estimate. If this study was omitted from the meta-analysis, the pooled OR estimate of the remaining was changed into 0.662 (95% CI was 0.491–0.894, $P<0.05$) using the fixed effects model ($Q=7.439$, $P=0.190>0.05$).

Another meta-analysis of six case–control studies was performed, and the pooled OR estimate was 0.645 (95% CI was 0.467–0.889, $P<0.05$) using the fixed effects model ($Q=10.123$, $P=0.072>0.05$). The Egger's test suggested that the publication bias did not exist (value of a was 1.618, $P=0.516>0.05$).

Meta-analysis of subgroup B

The pooled OR estimate for the odds of SLE in moderate alcohol drinking vs never or nearly never alcohol drinking for the seven studies was 0.723 (95% CI was 0.547–0.954, $P<0.05$) using the fixed effects model. Heterogeneity among them was tested without significance ($Q=9.983$, $P=0.125>0.05$). The funnel plot was shown in Fig. 2. The Egger's test suggested that the publication bias did not exist (value of a was 0.259, $P=0.900>0.05$). The sensitivity analysis found that the ORs of all the studies had no great influence on the estimate.

Table 2 The primary information of moderate alcohol drinking in meta-analysis

Author (reference)	Exposure vs un-exposure	OR (IRR) and 95% CI	Standard error[a]
Washio et al. [9]	Median duration: <5 years: 1–3 days/week vs <1 day/week	Median duration: <5 years: 2.33 (0.94–5.82)	0.46
	<10 years: 1–3 days/week vs <1 day/week	<10 years: 1.29 (0.60–2.77)	0.39
	Median duration: <5 years: 1–3 days/week vs <1 day/week	Median duration: <5 years: 0.60 (0.14–2.60)	0.74
	<10 years: 1–3 days/week vs <1 day/week	<10 years: 0.60 (0.16–2.22)	0.67
Bengtsson et al. [12]	(≤150 g/month and >150 g/month) vs 0 g/month	0.56 (0.33–0.96)	0.27
Ghaussy et al. [11]	3–5 drinks/week vs 0 drink/week	0.94 (0.28–3.17)[b]	0.62
Hardy et al. [5]	3–5 units vs 0 unit	0.41 (0.20–0.85)	0.37
Nagata et al. [6]	Weekly vs never	0.52 (0.25–1.06)	0.37
Formica et al. [10]	1–6 drinks/week vs 0 drinks/week	1.30 (0.70–2.40)	0.31

[a] Standard error was calculated based on the ORs and 95% CI.
[b] ORs with their 95% CI were calculated again based on the frequencies of alcohol drinking in the case and control groups.

Another meta-analysis of six case–control studies was performed, and the pooled OR estimate was 0.622 (95% CI was 0.456–0.850, $P<0.05$) using the fixed effects model ($Q=5.602$, $P=0.347>0.05$). The Egger's test suggested that the publication bias did not exist (value of a was 0.718, $P=0.605>0.05$).

Discussion

Conflicting evidence for the effect of moderate alcohol drinking on the development of SLE exists at present. In seven studies, four [6, 9–11] disclosed no association between alcohol drinking and SLE, whereas the other two [5, 12] reported that alcohol drinking may prevent the development of SLE. Only one study [9] found that high frequency drinkers showed an increased risk. Even in the same study, there might be different associations of alcohol drinking with SLE according to different alcohol consumptions.

In medical research, moderate and heavy alcohol consumption might have different effects on people's health actually. For example, moderate alcohol consumption was found to have a protective association with the incidence of chronic kidney diseases (CKD) [17], diabetes [18–20], and Graves' disease [21], and heavy drinking was found to increase the risk of CKD [22] or diabetes [18]. Additionally, high frequency drinkers might have felt emotional stress, which was a risk factor for SLE [7].

Moderate and heavy alcohol drinking was difficult to be separated clearly in all of the studies in our meta-analyses, so we had no way to perform subgroup analyses in all them with SLE. At present, the criteria of unit of alcohol consumption were different [15, 16, 23], and the criteria of moderate alcohol drinking were not same across countries [15, 16], too. Among the seven studies included in the meta-analyses, three were from Japan, two from America, one from England, and one from Sweden. Units of alcohol drinking used in those studies were different in

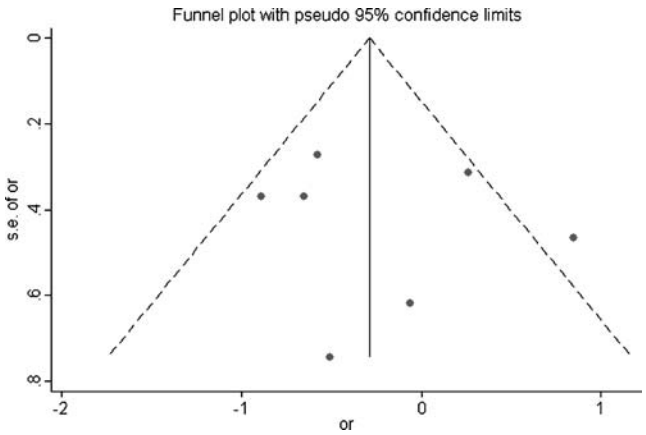

Fig. 1 Funnel plot of the odds ratio of SLE developing in moderate alcohol drinking of subgroup A compared with never or nearly never drinking in the six case–control studies and one cohort study

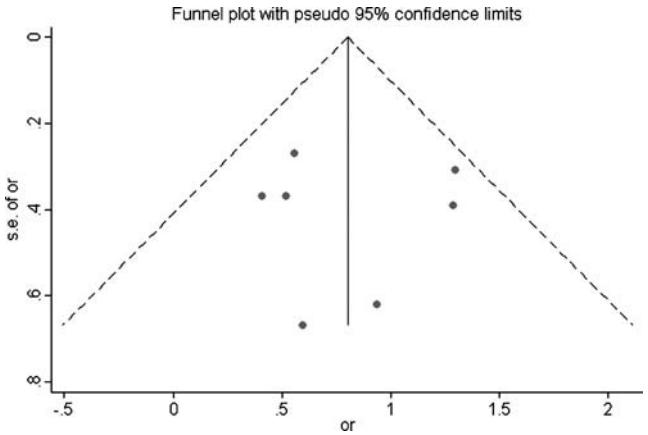

Fig. 2 Funnel plot of the odds ratio of SLE developing in moderate alcohol drinking of subgroup B compared with never or nearly never drinking in the six case–control studies and one cohort study

forms of day/week, drinks/week, grams/month, and units/week. We could redefine the definition of moderate alcohol drinking in the present paper based on those criteria [15, 16] mentioned above, which was relatively accordant, and only paid attention on the relationship of moderate alcohol drinking with SLE. Heavy alcohol drinking according to different countries and variance of individuals was too complicated to be redefined and analyzed.

The meta-analysis of subgroup A (including the patients treated for less than 5 years) found no association between moderate alcohol drinking and SLE (OR was 0.780 and 95% CI was 0.491–1.240, $P>0.05$), while the meta-analysis of subgroup B (including the patients treated for less than 10 years) provided that moderate alcohol drinking has a protective effect on the development of SLE (OR was 0.723 and 95% CI was 0.547–0.954, $P<0.05$). It was strange to find that although two meta-analyses were performed with different results, the values of ORs were close to each other. It was related to the effect models used in the meta-analyses. The ORs of moderate alcohol drinking of seven studies in subgroup A were heterogeneous; but those in subgroup B were homogeneous. Additionally, a sensitivity analysis was performed to find that the OR of each study had no great influence on the estimate in subgroup B and the OR of the study of Washio et al. had a great influence on the estimate in subgroup A. When the study of Washio et al. was omitted from the meta-analysis of subgroup A, the pooled OR estimate was change to 0.662 (95% CI was 0.491–0.894, $P<0.05$), which suggested that moderate alcohol drinking had a protective effect on the development of SLE.

The pooled estimates of six case–control studies in subgroups A and B were comparable ($0.645>0.622$) because they all were calculated through the fixed effects model with significance, which suggested that the protective effect of moderate alcohol drinking on SLE was related to the duration of SLE patients. The longer the duration of SLE, the larger protective effect moderate alcohol drinking had on SLE.

The publication bias was tested by Egger's test without existence in the two subgroups' meta-analyses. The absence of publication bias was probably due to the fact that the investigators paid little attention on the association of alcohol drinking with the development of SLE in their studies. All the information provided that the results of the meta-analyses were reliable.

Although the biologic pathway through which moderate alcohol drinking acts to decrease the risk of SLE is unclear, explanations were suggested by some researchers. It is possible that the cardioprotective effects of moderate alcohol intake [19, 21] and the mechanisms leading to these effects might well be acting in the vasculature of patients with SLE [24]. Kannel and Ellison emphasized the well-documented increased risk of cardiovascular mortality presented by excess drinking, citing a U-shaped mortality curve cast by the combined protective and harmful influences of alcohol [25]. The primary symptom of SLE was renal dysfunction. Schaeffner et al. [17] deduced in their study that an alcohol-related increase in high density lipoprotein cholesterol may explain the potential beneficial effect on renal dysfunction, which could explain its beneficial effects on SLE.

There are several limitations in our study. In those reviewed studies in our meta-analyses, there inevitably existed variations in the aspects that followed: (1) certain genetic factors would have influence on the development of SLE according to different ethnic groups; (2) subjects investigated in those studies were different; some included men and women SLE and the others only included women SLE; (3) SLE patients were newly diagnosed in some studies and not in other studies; (4) populations from which controls were randomly selected were different in case–control studies; (5) the definition of alcohol drinking was different in those studies; moreover, the situation of alcohol drinking of the same person might have changed before and after the development of SLE; (6) social class is not only strongly linked to alcohol and cigarette consumption, but also to health care access and speed of diagnosis of SLE. It is likely that wealthier people, who drink less alcohol, have better access to health care and are more likely to be diagnosed promptly with SLE; and (7) the confounders and effect modifiers for the relationship between alcohol drinking and SLE were different in these studies and other important confounders were not taken into account; in addition, some ORs with their 95% CI were calculated again based on the raw data, which had not taken confounders into account at all.

In spite of these limitations, we performed some measurement especially in the aspect of definition of moderate alcohol drinking. We redefined it based on the criteria of different countries in order that the definition of moderate alcohol drinking of different studies was in accordance with each other to the full. At the same time, nearly all the ORs of moderate alcohol drinking vs never drinking were adopted or calculated again. Only two studies presented the ORs of moderate alcohol drinking vs drinking of less than 1 day/week. Additionally, the heterogeneity test was performed and the random effects model was used to obtain the pooled OR estimate with consideration of the heterogeneity among those studies in the meta-analyses.

As might be expected, future studies of a large scale, especially cohort studies of different genetic background, could contribute to the knowledge of the association between moderate alcohol drinking and SLE, especially the association of heavy drinking with SLE. Additionally, in vitro and animal studies may further help to shed light on

the biologic mechanisms and pathways by which alcohol drinking may play a role in the etiology of SLE.

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Disclosures None.

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