REVIEW

CORRELATION BETWEEN ALCOHOL CONSUMPTION AND MYOCARDIAL INFARCTION: DOSE-RESPONSE META-ANALYSIS OF 18 COHORT STUDIES

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Abstract

It was demonstrated that alcohol has double effect on myocardial infarction (MI). The results of the studies that investigated the correlation between alcohol drinking and MI are varied. The aim of the present meta-analysis is to combine current prospective cohort studies of alcohol consumption and MI and to analyse the potential dose-response relationship. A complete search on Medline and Embase databases from database creation to May, 2016 was performed. We included in the meta-analysis only prospective cohort studies in English: these containing drinking doses divided into three or more levels and studies that contain relative risk/hazard ratio (RR/HR) and 95% confidence interval (CI) of MI onset risk, or adequate data for calculation of these indexes. RR of the alcohol consumption group, low-dose alcohol consumption group, moderate alcohol consumption group and high-dose alcohol consumption group were 0.770 (95% CI 0.681 - 0.871), 0.861 (95% CI 0.786 - 0.942), 0.721 (95% CI 0.609 - 0.853) and 0.620 (95% CI 0.484 - 0.794) respectively. According to districts, follow-up periods and sample sizes, subgroups were divided and in all subgroups it was seen that alcohol consumption could reduce the onset risk of MI. When the drinking dose was lower than 96 g/day, dose meta-analysis showed that the linear test result $p < 0.001$; thus the drinking dose and MI onset risk were in non-linear negative correlation. With the increase of drinking dose, the onset risk of MI declined. Sensitivity analysis indicated stable study results; no publication bias showed in begg's test and egger's test. In conclusion, there is a negative correlation between drinking doses and MI; with the increase of drinking doses, the incidence rate of MI decreases.

Rezumat

S-a demonstrat faptul că alcoolul are dublă acțiune în infarctul de miocard (IM). Rezultatele studiilor care investighează legătura dintre consumul de alcool și IM sunt variate. Scopul prezentei meta-analize este de a combina studiile de cohort actuale cu privire la consumul de alcool și IM și să analizeze potențiala legătură doză-efect. S-a realizat o căutare completă în bazele de date Medline and Embase de la crearea bazelor de date și până în mai 2016. În meta-analiză au fost incluse numai studiile prospective de cohortă scrise în limba engleză, care conțin dozele de alcool consumate, divizate pe 3 sau mai multe nivele și studiile care conțin valorile riscului relativ (RR) și 95% interval de confidențialitate (IC) pentru IM, or date adecvate care permit calcularea acestor indici. RR pentru grupurile de consumatori de alcool au fost 0,770 (95% CI 0,681 - 0,871), 0,861 (95% CI 0,786 - 0,942), 0,721 (95% CI 0,609 - 0,853) și respectiv, 0,620 (95% CI 0,484 - 0,794) pentru grupul consumator de doze mici de alcool, consumator de doze moderate de alcool, respectiv, consumator de doze mari de alcool. Grupurile au fost subdivizate în funcție de regiune, perioada de urmărire și mărimea grupului, însă în toate subgrupurile s-a observant faptul că riscul de apariție a IM este redus de consumul de alcool. Când dozele de alcool au fost mai mici de 96g/zi, meta-analizele au arătat că rezultatele testului linear au prezentat un $p < 0,001$, ceea ce arată că dozele de alcool și riscul de apariție a IM au fost în corelație negativă non-liniară. Odată cu creșterea dozelor, riscul de apariție a IM a scăzut. Analiza sensibilității indică stabilitatea rezultatelor obținute; nici un studiu nu a arătat modificări în testele *begg* și *egger*. În concluzie, există o legătură negativă între consumul de alcool și IM: odată cu creșterea dozelor, riscul de IM scade.

Keywords: myocardial infarction; alcohol consumption; dose-response meta-analysis

Introduction

Myocardial infarction (MI) usually appears in middle aged and elderly people. It is characterized by acute onset, high disability and fatality rates. 2 million people die every year from acute MI, and this become a threat to global public health and an important factor that increases burden of diseases and death worldwide [49]. Taking all these in consideration it is very important to understand the risk factors of MI. The pathogenesis of MI is complicated and many factors have both protective and damage effects, from which the alcohol

consumption is the most controversial one [8, 10, 12, 39, 47, 53]. Changes in the drinking doses can change the protective and damage effects.

In recent decades, many studies were carried out to investigate the correlation between alcohol consumption and MI and results were varied. Some studies indicated that moderate drinking could reduce the incidence rate of MI and the correlation between alcohol consumption and MI was U-shaped and J-shaped [2, 23, 40, 46]. However, alcohol consumption and MI were in negative correlation [14] in some studies and positive correlation [26, 44] in others. The aim of this study is to collect all prospective cohort studies of alcohol consumption and MI onset risks and to clarify the potential relationship between these two.

Materials and Methods

Literatures in Medline and Embase databases that might be related to alcohol consumption and MI were searched and the retrieval time was from database creation to May, 2016. In the meantime, references of relevant reviews and meta-analyses were back traced. Index words included myocardial infarction [MESH]; Myocardial Infarct* OR MI, alcohol [MESH]; Alcohol Drinking OR Drinking; Alcohol OR Alcohol Consumption; Alcohol OR ethanol; cohort studies [MESH].

Inclusion criteria. Literatures meeting the following requirements were included in the meta-analysis: published English prospective cohort studies; drinking doses were grouped and divided into three or more levels; subjects were followed up after alcohol drinking to observe MI conditions; studies containing relative risk/hazard ratio (RR/HR) of different drinking doses and MI onset risks or adequate data for calculation of these indexes.

Exclusion criteria. Review literatures, non-human researches and literatures containing inadequate data and repeated reports were excluded. Only studies with the most complete samples were used.

Data extraction and quality evaluation of the studies. Data were extracted by two researchers according to the same standards. A third researcher would participate in discussion if disagreements occurred. Extracting information included the first author, publication year, age, gender, country, follow-up visit time, sample size, drinking dose, RR/HR (95% confidence interval) or the number of incidence (the overall number of people) and correction concomitant variables of multiple analysis. According to that 1 glass of wine = 12.5 g and 1 mL = 0.8 g, the drinking quantity was converted to ethyl alcohol consumption (g/day). Thus the low, medium and high doses of drinking were $\leq$ 12.5 g/day, 12.5 - 50 g/day and $\geq$ 50 g/day respectively. In the extraction of doses, the mid-value of upper limit and lower limit was taken as the drinking dose. If the upper limit was open, the drinking quantity was 1.2 times that of the lower limit. The reference group was the non-drinking group or lowest-dose group; if the reference group was not the lowest-dose group, the conversion was based on the excel table provided by the website http://www.pnlee.co.uk/software.htm. Quality of the studies was evaluated by Newcastle-Ottawa scale: 0 - 3 scores indicated low quality; 4 - 6 scores indicated medium quality and 7 - 9 scores indicated high quality.

Statistical analysis. Suppose HR values in reference reports were equal to RR, medium and low-dose drinking and the MI onset risks of objects in the reference group were calculated together; heterogeneity tests were evaluated through q-test and I^2 statistics [18]. $p < 0.05$ and $I^2 > 50\%$ indicated high heterogeneity. Random effects model of DerSimonian and Laird was used to combine effect sizes; otherwise, $p < 0.05$ or $I^2 > 50\%$, and fixed effect model was used [13, 34]. Harbord test [16] and begger [4] tests were used to reflect publication bias. Subgroup analysis was based on districts, follow-up time and sample sizes. The correlation between drinking doses and MI was explored by dose-response meta-analysis [41]. Stata13 was used for statistical analysis of all data.

Results and Discussion

After data collection, there were totally 6,048 articles that might contain relevant data. After screening based on titles and abstracts, 5,950 studies were excluded. Then contents of the remaining articles were read and 83 articles were excluded. After that, 3 literatures were added after reading relevant literatures. Therefore, this meta-analysis totally included 18 studies [3, 6, 9, 12,14, 21, 22, 26, 27, 28, 29, 30, 33, 36, 37, 44, 50, 54] (Figure 1).

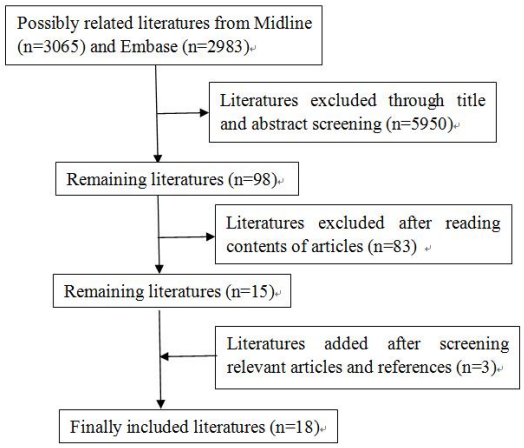

Figure 1.

Flow chart illustrating the literature search for 18 cohort studies on alcohol consumption and MI risks

In addition, one study was based on different districts; one study was based on different genders and another was based on lethality. Therefore, there were totally 21 studies, including 956,151 people. Studies included in this meta-analysis were: 5 American, 4 Asian, 8 European and 1 containing multiple centres. 3 studies contained both males and females, 1 study contained only females and 14 studies contained only males; 11 studies contained sample sizes bigger than 10 thousand and 7 studies contained samples sizes smaller than 10 thousand; 9 was follow-up studies longer than 10 years and 9 were follow-up studies on a period less than 10 years; 8 studies had high quality, 10 had medium quality and no study was in low quality. Basic characteristics of studies are shown in Table I.

Table I

Basic characteristics of 18 prospective cohort studies of drinking and MI onset risks

Study	Countries	Gender	Age (years)	Disease	follow-up (years)	No. of levels	n	RR or Events (Total)	Concomitant variable	NOS score
Kozarevic D. (1982)	Tuzla, Bosnia, Remetinec, Croatia	male	Born 1903 - 1928 Recruit 1964 - 1965	MI	7	0 0 - 0.25 0.25 - 1 1 - 2 >2 (drinks/week)	10558	15 (1311) 11 (1167) 26 (4621) 8 (2490) 4 (969)	None available	4
Kittner S.J. (1983)	Puerto Rican	male	35 - 79	MI	8	0 1 - 14 15 - 39 40 - 79 > 80 (g/daily)	9150	133 (6983) 7 (496) 9 (895) 6 (433) 5 (343)	Age, number of cigarettes smoked, exercise, urban/rural status, income	4
Kauhanen J. (1997)	Kuopio, Finland, or surrounding rural communities	male	42,48,54,or 60	AMI	7.7	> 3 bottles of beer/session 3 - 6 bottles of beer/session < 6 bottles of beer/session	1641	1 1.28 (0.85 - 1.93) 0.96 (0.39 - 2.35)	Age, total alcohol, body mass index, fibrinogen, low and high density lipoprotein cholesterol, systolic blood pressure, previous diseases, occupational status, marital status, involvement in organizations, smoking, leisure time physical activity, depression	5
				MI				1 2.40 (0.95 - 6.06) 7.05 (1.93 - 25.67)		
Camargo C.A. Jr. (1997)	America	male	40 - 84	MI	11	< 1 0.14 0.14 - 0.57 0.57 - 1 1 1 - 2 (drinks/daily)	21530	1 1.08 (0.85 - 1.38) 0.96 (0.78 - 1.20) 0.82 (0.62 - 1.07) 0.65 (0.52 - 0.81) 0.53 (0.32 - 0.88)	Age, aspirin use, β-carotene use, smoking, exercise, diabetes mellitus, parental history of MI	7
Yano K. (1997)	Japan	male	born: 1900 - 1919 ricruit: 1965 - 1968	MI	6	0 1 - 6 7 - 15 16 - 39 > 40 (mL/day)	7591	76 (3565) 23 (1034) 13 (962) 16 (1024) 4 (1006)	Age	5
Kitamura A. (1998)	Japan	male	40 - 59	MI	8.8	0 1 - 22 23 - 45 46 - 68 69 - (g/day)	8483	1 0.85 (0.39 - 1.86) 0.57 (0.25 - 1.31) 0.43 (0.16 - 1.16) 0.65 (0.20 - 2.12)	Age, serum total cholesterol, cigarette smoking, BMI, left Ventricular hypertrophy, history of diabetes mellitus	6
Mukamal K.J. (2003)	America	male	40 - 75	MI	12	0 0 - 4.9 5.0 - 9.9 10.0 - 14.9 15.0 - 29.9 30.0 - 49.9 ≥ 50.0	38077	1 0.98 (0.84 - 1.15) 0.83 (0.68 - 1.00) 0.69 (0.57 - 0.85) 0.79 (0.64 - 0.96) 0.64 (0.51 - 0.80) 0.48 (0.33 - 0.69)	Age, smoking, BMI, diabetes, energy intake, vitamin E, fat, dietary fiber	8
Marques-Vidal P. (2004)	France	male	50 - 59	MI FMI	5	0 0 - 18 18 - 38 38 - 63 > 63 (mL/daily)	7352	1 0.59 (0.30 - 1.17) 0.50 (0.25 - 1.0) 0.42 (0.21 - 0.85) 0.27 (0.13 - 0.59)	Age, marital status, educational level, vigorous exercise, BMI, blood pressure, diastolic blood pressure, total cholesterol, tri-glycerides, smoking status, anti-hyper-tensive drug treatment and hypolipidemic drug treatment	6
	Ireland					0 0 - 16 16 - 32 32 - 59 > 59 (mL/daily)	2398	1 0.61 (0.26 - 1.84) 0.68 (0.31 - 1.47) 0.57 (0.25 - 1.30) 0.43 (0.17 - 1.09)	Age, marital status, educational level, vigorous exercise, BMI, blood pressure, diastolic blood pressure, total cholesterol, tri-glycerides, smoking status, anti-hyper-tensive drug treatment and hypolipidemic drug treatment	

Study	Countries	Gender	Age (years)	Disease	follow-up (years)	No. of levels	n	RR or Events (Total)	Concomitant variable	NOS score
Beulens J.W. (2007)	America	male	40 - 75	MI	16	0 0.1 - 4.9 5.0 - 9.9 10.0 - 14.9 15 - 29.9 30 - 49.9 50 – (g/d)	11711	1 1.09 (0.86 - 1.37) 0.81 (0.60 - 1.08) 0.68 (0.51 - 0.91) 0.72 (0.54 - 0.97) 0.67 (0.48 - 0.94) 0.41 (0.22 - 0.77)	smoking, BMI, physical activity, hypercholesterolemia, family history of MI, aspirin use, lipid-lowering therapy, energy intake, energy-adjusted quintiles of saturated fat, trans fatty acids, sodium, potassium, magnesium, folate, vitamin E, ω-3 fatty acids, dietary fiber	7
Bazzano L.A. (2009)	Chinese	male	≥ 40	MI FMI	> 8	0 1 1 - 5 > 5 (drinks/daily)	494084	1 0.93 (0.70 - 1.24) 0.66 (0.54 - 0.82) 0.58 (0.41 - 0.81)	Age, cigarette smoking, physical activity, BMI, systolic blood pressure, education, geographic region, urbanization, self-reported history of diabetes	6
Chiuve S.E. (2010)	America	female	30 - 55	MI	≥ 26	0 former 0.1 - 4.9 5.0 - 14.9 15.0 - 29.9 > 30(g/day)	85067	1 0.95 (0.84 - 1.08) 0.94 (0.84 - 1.05) 0.79 (0.69 - 0.91) 0.63 (0.50 - 0.79) 0.59 (0.46 - 0.75)	Age, calories, smoking, BMI, parental history of MI, menopausal status, use of postmenopausal hormones, aspirin use, multivitamin and vitamin E supplements, physical activity and intake of marine omega-3fat, alpha-linolenic fat, trans fat, ratio of polyunsaturated to saturated fat, diagnosis of stroke, diabetes, high blood pressure or high cholesterol	7
Merry A.H. (2011)	Maastricht and surrounding	both	20 - 59	AMI	11.1	0 0 - 2 2 - 4 > 4 (glasses/day)	21148	1 0.69 (0.50 - 0.94) 0.61 (0.42 - 0.90) 0.54 (0.34 - 0.86)	Age, sex, baseline cohort, smoking status, smoking frequency and duration, total cholesterol level, diabetes mellitus, education, family history of MI, BMI	7
Ilomaki J. (2012)	Kuopio	male	42, 48, 54, 60	MI	12	< 1.8 1.8 - 12 12 - 24 > 24 (g/day)	1030	1 0.79 (0.53 - 1.16) 0.95 (0.58 - 1.51) 1.23 (0.67 - 2 14)	Age, smoking, working status, cigarette years, BMI, HDL C, systolic blood pressure, insulin, fibrinogen, history of CVD	6
Makita S. (2012)	Japan	male	40 - 80	MI	5.5	0 17.4 49.7 (g/day)	8059	1 0.49 (0.24 - 0.98) 0.53 (0.25 - 1.12)	Age, hypertension, diabetes, dyslipidemia, smoking index, BMI	6
Romelsjo A. (2012)	Sweden	male	18 - 20	MI FMI	35	0 0.1 - 10 10 - 30 30 - 60 > 60 (g/day)	48709	1 1.44 (1.05 - 1.98) 1.83 (1.32 - 2.53) 1.96 (1.26 - 3.05) 1.61 (0.85 - 3.06)	Smoking, works, divorced parents, run away from home, trance, low emotional control, low social maturity, Moderate IQ, Low IQ, fair health, BMI	6
Gémes K. (2015)	Norway	both	≥ 20	AMI	11.6	0 0 - 0.36, 0.36 - 0.7, 0.7 - 1 > 1 problem drinkers (drinks/day)	58827	1 0.88 (0.78 - 0.99) 0.75 (0.65 - 0.86) 0.84 (0.66 - 1.01) 0.70 (0.51 - 0.97) 0.84 (0.65 - 1.08)	Age, sex, level of education, cohabiting, smoking, physical activity and BMI	8
Kuanrong Li (2014)	Heidelberg	male	< 61.5 ≥ 61.5	AMI	11.4	< 10 10 - 30 > 30 < 10 10 - 30 > 30	10981	1 0.93 (0.64 - 1.35) 0.94 (0.64 - 1.38) 1 0.82 (0.58 - 1.15) 0.58 (0.39 - 0.86)	Smoking status, BMI, physical activity and educational	7
Smyth A. (2015)	12 countries	both	35 - 70	MI FMI	4.3	0 0 - 1 female 1 - 2 or men 1 - 3 more (drinks/day)	109755	1 0.77 (0.63 - 0.94) 0.65 (0.44 - 0.97) 0.78 (0.56 – 1.09)	Age, sex, smoking, ethnicity, education, body-mass index, diabetes, hypertension, jaundice and hepatitis, physical activity, diet, medications, wealth index, total alcohol consumption and heavy episodic	7

*BMI = body mass index; CVD = cardiovascular diseases; HDL-C = high density low cholesterol

Results of meta-analysis. Compared with the non-alcohol consumption group and low alcohol consumption group, different doses of drinking could lower the onset risks of MI. The MI onset risk of drinking group was RR = 0.770 (95% CI, 0.681 - 0.871, I^2 = 81.0%, p < 0.001) (Figure 2). The MI onset risk of

low-dose drinking group was RR = 0.861 (95% CI, 0.786 - 0.942, I^2 = 35.4%, p < 0.069). The MI onset risk of medium-dose drinking group was RR = 0.721 (95% CI, 0.609 - 0.853, I^2 = 77.6%, p < 0.001). The MI onset risk of high-dose drinking group was RR = 0.620 (95% CI, 0.484 - 0.794, I^2 = 74.3%, p < 0.001).

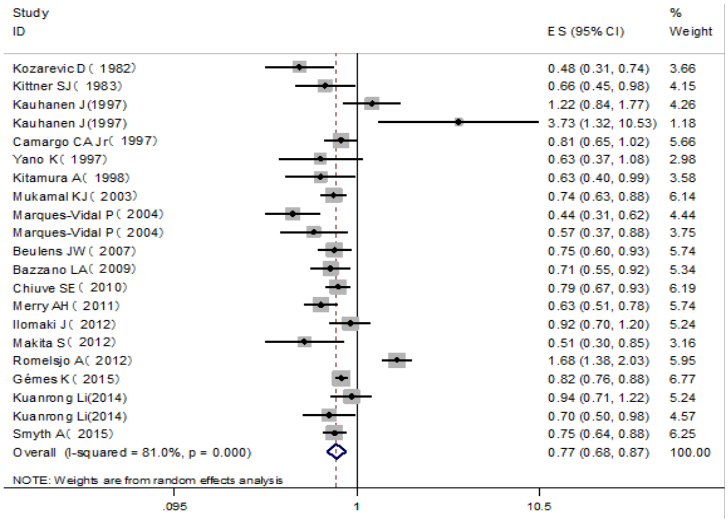

Figure 2.
Forest plot of RR with CI for alcohol consumption and myocardial infarction risk

Results of subgroup analysis. Subgroup analyses were carried out according to different districts, follow-up time and sample sizes and MI incidence rates of all subgroups decreased due to alcohol consumption (Table II). Subgroup analyses showed that, in Asia, Europe and America, different doses of alcohol consumption could lower the onset risk of MI. However, the protective effect of drinking was more significant in Asians; RR values of medium and high doses were lower; RR values of studies with less than 10 years of follow-up visits were lower and RR values of researches with sample sizes ≥ 10,000 and < 10,000 were similar.

Table II
Subgroup analyses of the association between alcohol consumption and myocardial infarction risk

Sample		Follow-up		Areas				
< 10,000	≥ 10,000	≥ 10 years	< 10 years	Asia	America	Europe	Parameter	Subgroup
9	12	10	11	4	5	11	n	Reference *vs.* Alcohol consumption
74.8	84.5	85.7	68.5	0	0	89.3	12 (%)	
< 0.001	< 0.001	< 0.001	< 0.001	0.704	0.897	< 0.001	p	
0.770 (0.681 - 0.871)	0.797 (0.693 - 0.917)	0.847 (0.724 - 0.990)	0.680 (0.555 - 0.832)	0.653 (0.539 - 0.792)	0.763 (0.696 - 0.836)	0.833 (0.658 - 1.055)	RR (95% CI)	
8	10	8	10	3	5	9	n	Reference *vs.* Low-dose drinking
29.9	43.0	45.7	32.9	36.8	0	67.7	12 (%)	
0.190	0.071	0.0075	0.145	0.064	0.994	0.002	p	
0.905 (0.701 - 1.169)	0.850 (0.774 - 0.935)	0.864 (0.776 - 0.961)	0.856 (0.712 - 1.031)	0.901 (0.712 - 1.139)	0.851 (0.768 - 0.944)	0.897 (0.715 - 1.125)	RR (95% CI)	
9	12	10	11	4	5	11	n	Reference *vs.* Medium-dose drinking
66.0	83.3	85.5	54.4	0	0	84.6	12 (%)	
0.003	< 0.001	< 0.001	0.016	0.710	0.602	< 0.001	p	
0.718 (0.491 - 1.050)	0.729 (0.600 - 0.886)	0.793 (0.631 - 0.996)	0.622 (0.487 - 0.795)	0.627 (0.520 - 0.755)	0.652 (0.585 - 0.726)	0.836 (0.602 - 1.160)	RR (95% CI)	
4	8	6	6	2	4	6	n	Reference *vs.* Medium and high-dose drinking
31.1	77.8	79.9	4.5	0	83.0	72.4	12 (%)	
0.226	< 0.001	< 0.001	0.388	< 0.762	0.001	0.003	p	
0.480 (0.306 - 0.751)	0.678 (0.516 - 0.892)	0.724 (0.533 - 0.984)	0.523 (0.405 - 0.676)	0.568 (0.416 - 0.774)	0.633 (0.399 - 1.004)	0.608 (0.383 - 0.963)	RR (95% CI)	

Results of dose-response analysis. Five studies could not provide sufficient details, thus there were 13 studies included for dose-response analysis. The maximum alcohol consumption dose in this study was 96 g/day and the linear test result $p < 0.001$, indicating that there was a non-linear relationship between drinking doses and the incidence rate of MI. The dose-response figure showed that, with the increase of drinking doses, the onset risk of MI decreased continually. When the dose was less than 10 g/day, RR declined rapidly; when the dose was between 10 and 35 g/day, RR was relatively stable; when the dose > 35 g/day, RR showed a decreasing tendency again (Figure 3).

Sensitivity analysis and publication bias. Partial researches were eliminated and the rest researches were combined. Results showed no significant changes, indicating the meta-analysis results were relatively stable (Figure 4). Begg's test Z = 1.00, p = 0.319; egger's test t = -0.65, p = 0.521 (-2.654 - 1.391) and no publication bias was found.

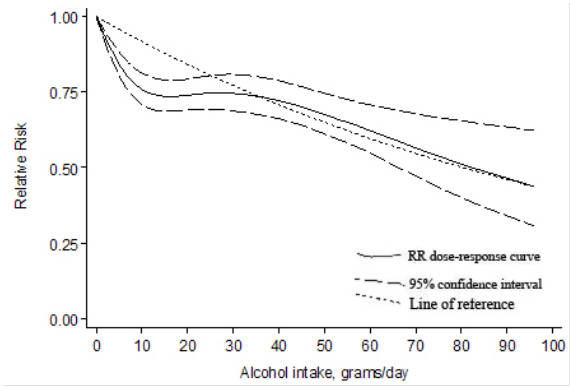

Figure 3.
The dose-response analysis between alcohol consumption and myocardial infarction risk

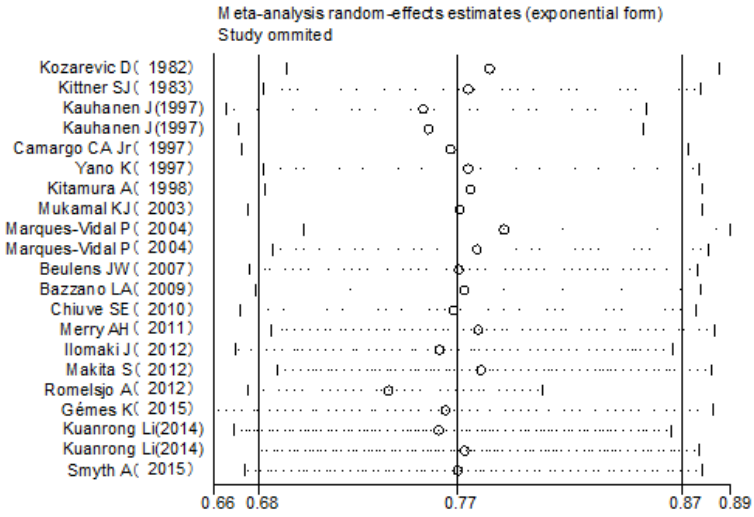

Figure 4.
Sensitivity analysis of the 21 Cohort Studies

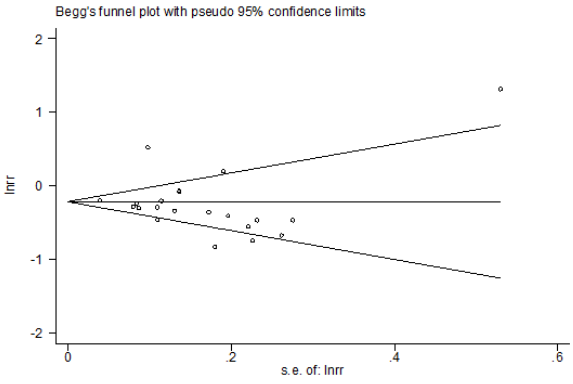

Figure 5.
Begg's Funnel Plot of the 21 Cohort Studies

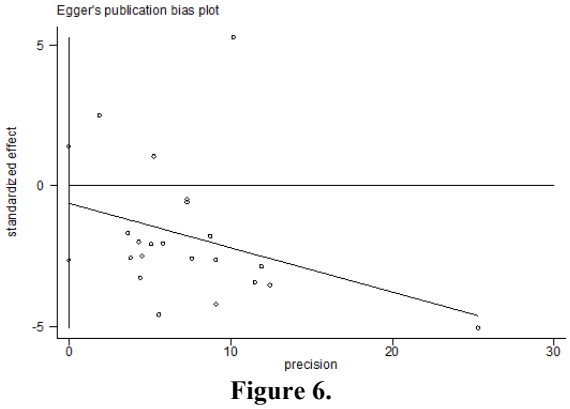

Figure 6.
Egger's Publication Bias Plot of the 21 Cohort Studies

On the basis of cohort studies, this study discussed the potential relationship between alcohol consumption doses and onset risks of MI. Meta-analysis results

showed that different alcohol consumption doses could all lower the onset risk of MI. Results of dose-response meta-analysis indicated the negative correlation between drinking doses and MI onset risk: with the increase of drinking doses, the onset risk of MI decreased continually. Subgroup analyses based on different areas, follow-up time and sample sizes also showed a decreasing tendency of MI onset risks. Recently published meta-analyses mainly aimed at the relationship between alcohol consumption and coronary heart disease (CHD) and cardiac vascular disease (CVD). Liu [31], Ronksley [45] and Zhang [55] studied the relationship between alcohol consumption and CHD onset risks through 15, 29 and 18 cohort studies respectively; results indicated that different drinking doses could all lower the onset risk of CHD. Liu carried out researches on alcohol consumption of East Asian males, which showed more significant decreasing effect than that of researches carried out by Ronksley and Zhang. Such result was similar to this study. In this study, the protective effect of alcohol consumption on MI onset risks of Asians was more significant compared with that on Europeans and Americans. Huang [19] analysed the relationship between alcohol consumption of hypertensive patients and CVD through 9 studies (11 cohort studies); results showed that different doses of alcohol consumption could also reduce the onset risk of CVD of hypertensive patients. However, because research objects were hypertensive patients, doses of alcohol consumption were low (the maximum dose was 30 g/day). Corrao [11] analysed the relationship between alcohol consumption and CHD onset through 28 high-quality cohort studies and found that there was a J-shaped relationship between alcohol consumption and CHD. However, relevant studies of drinking and CHD death were also included in the meta-analysis. Thus the J-shaped relationship might be due to death rates caused by cancer, liver cirrhosis and diabetes because of drinking, which was similar to the all-cause mortality results in the research carried out by Huang [39]. Moreover, Mostofsky [13] found that 24 h after a moderate amount of alcohol consumption, risks of CVD increased; as time passed, the risks decreased; risks of long-term follow-up visits were not studied. Therefore, above meta-analyses indicated that long-term regular alcohol consumption could reduce risks of CVD and CHD; however, death rates of other relevant diseases could be increased. This study mainly analysed the correlation between alcohol consumption and MI onset risks. It was found that long-term regular alcohol consumption had protective effect on MI.

On the basis of atherosclerosis, MI is caused by myocardial anoxia and necrosis due to blood coagulation and blocking inside blood vessels due to the bleeding of plaque rapture. Alcohol consumption can have great influence on MI, but its relationship with MI pathogenesis is controversial. The first one is the protective effect of alcohol consumption. Most researches showed that alcohol consumption could increase high density lipoprotein cholesterol (HDLC), apolipoprotein A1 and adiponectin and reduce fibrinogen level, in which the HDLC was the main protective factor which could explain about 50% of the causal relationship [5, 24, 42, 25]. However, some researchers believed that the value of HDLC was low, which could only explain about 16% of the causal relationship [32-35]. Besides, some researches found that alcohol consumption could increase the prostacyclin of blood vessel wall, improve functions of vascular endothelial cells, increase insulin sensitivity and resist thrombosis [7, 38]; moreover, long-term regular alcohol consumption could improve heart rate variability (HRV) [20, 43] and thus reduce MI onset risks. The second one is the damage effect of alcohol consumption. Most researches showed that alcohol consumption could increase LDHC, triglyceride, heart rate, blood pressure [1, 48, 51] and thus increase the risks of atherosclerosis, atrial fibrillation and anoxia, resulting in damaging cardiac muscle cells and cardiovascular system and producing fibrinolytic enzyme inhibitor [17]. Stenestrand founds that the increase of blood pressure within a certain range could reduce the death rate of ICU patients with chest pain [52]. Brien SE discovered that alcohol consumption had no significance to LDHC and triglyceride but could mainly affect high-density hydrocarbons (HDHC) adiponectin and fibrinogen level [7]. Thus it could be seen that the mechanism between alcohol consumption and angiocarpy was extremely complicated and current paradoxes were various. The relationship between alcohol drinking and MI was mainly explored through epidemiological investigations and studies.

This study had two advantages. First of all, we analysed MI systematically and comprehensively; MI diagnosis was clear and misclassifications were few, thus the obtained results had high authenticity. Secondly, we used prospective cohort studies and inherent selection bias and recalling bias were eliminated; causality validation was powerful, latest researches were added and sample sizes were enlarged, thus the results were more reliable and stable.

In the meantime, this study also contained certain limitations. First of all, doses of alcohol consumption were obtained through self-administered questionnaires, thus there might be errors. Secondly, included contents about females were insufficient while there were more and more females began to drink alcohol, thus there was certain gender bias. Thirdly, positive results were few, which could lead to over-estimation of the protective effect of alcohol consumption, or positive results were difficult to be published or authors considered them insignificant.

Fourthly, different studies had different alcohol consumption classification rules, which might cause errors. Fifthly, only English studies were included. Sixthly, there was only one research carried out on the relationship between alcohol consumption of teenagers and MI. Thus more studies on alcohol consumption of teenagers and the lethality of MI should be done in the future. Seventhly, studies without detailed information were eliminated from the dose-response analysis, which could result in bias.

Conclusions

In conclusion, we retrieve prospective cohort studies of alcohol consumption and MI onset and results are merged. It is found that doses of alcohol consumption are in negative correlation with MI onset risks. However, more samples and prospective cohort studies are needed in the future to further clarify the correlation between these two, thus to better prevent MI.

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