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The Role of Alcohol Consumption in Cardiovascular Health: A Systematic Review and Meta-analysis

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

There remains a debate on the effects of alcohol use on cardiovascular health. This systematic review and meta-analysis examined the relationship between this link and blood pressure, lipid profiles, and the frequency of cardiovascular events to shed light on it. A comprehensive search strategy using PRISMA guidelines resulted in nine papers that met the inclusion requirements. Moderate alcohol usage was shown to have a U- or J-shaped connection with cardiovascular events; excessive intake or abstinence was associated with increased risk, but moderate consumption had preventive advantages. Moderate drinkers showed positive changes in their lipid profiles and lowered blood pressure in comparison to heavy or non-drinkers. The wide range of research highlights the need to consider individual characteristics and study techniques. Despite the potential cardiovascular benefits of moderate alcohol use shown by these studies, treatment recommendations should be cautious. To provide personalized recommendations to patients and policymakers, future research should focus on explaining mechanisms, examining modifiers, and assessing the effects of different types of alcohol and drinking habits on cardiovascular health.

Keywords: Alcohol consumption; cardiovascular health; mental health; patients' quality of life.

1. INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of premature mortality, presumed to contribute the highest number of lost life years on a global scale [1]. In fact, deaths from CVD outnumber in aggregate all of the other most significant causes of death (cancer, infectious diseases & eating disorders combined) by a rate nearly double. CVD is the leading cause of morbidity and also significantly affects patients' quality of life [2,3]. The most common forms of CVD are coronary heart disease (CHD) or cerebrovascular disease.

A main risk factor for CVD, is alcohol intake. Alcohol use is associated with more than 200 diseases and injuries and nearly 3 million deaths each year. The relationship between alcohol consumption and cardiovascular events also follows a well-known U- or J-shaped curve [4,5]. What seems clear, though, is that moderate alcohol intake has a protective effect with respect to CHD probably through mechanisms like elevation of HDL cholesterol and an inhibition of early stage of atherosclerosis. Heavy drinking raises the risk of cardiovascular events [6]. The range that offered the most risk was between 38 g per day or 23 drinks per week. Moreover, research indicates that whereas heavy drinking is connected to an exponential rise in illness, moderate drinking is associated with decreased risk of cardiovascular disease [7].

Academics disagree on the hypothesis that alcohol use might protect against a number of diseases. From a pharmacological point of view, alcohol use interacts with several medications, including diuretics, antidepressants, and opioids [8], since it can change the way that pharmaceuticals and/or alcohol are metabolized [9]. Lower alcohol metabolism can result in greater blood alcohol levels, and this can be caused by histamine H2 receptors and other drugs used to treat heartburn and ulcers [9]. It is necessary to use caution while taking resveratrol with some medicines, such as those found in wine, as alcohol might alter a drug's metabolism [10].

Alcohol consumption is particularly detrimental to mental health since it increases the risk of suicide when used acutely and in large doses [11]. In conclusion, from the standpoint of a cardiologist, studies conducted over the years have suggested that wine and mild to moderate alcohol use may help reduce the risk of cardiovascular disease [12,13-16]. Recently, studies that have used Mendelian randomization approaches have questioned this impact. Reduced alcohol consumption is beneficial for cardiovascular health, according to the genetic approach analyses of these studies, which revealed that people with the alcohol dehydrogenase 1B (ADH1B) gene had a decreased risk of coronary heart disease when they drank less alcohol [17]. However, research has not been broken down into categories of alcoholic beverages [18].

Reduced risk of breast cancer is observed in women who consume up to one drink per day [18], and an inverse relationship between an acute intake of alcohol with ischemic heart disease incidence as well as the prognosis for patients under risk of future coronary episodes has been reported from several studies [19]. For example, consuming anything between 0 to 7.5 drinks per week, or less than 12.5 grams per day is the least harmful for health, as against the greater risk associated with a consumption over 38 grams per day or more than 23 drinks in a week [7]. Nevertheless, the favorable effects of alcohol on heart health must be balanced with its risks; heavy drinking is associated with an increasingly raised risk for both CVD and other health problems.

Even more concerning, the interplay between alcohol and many medications such as diuretics, antidepressants, and opioids complicates treatment plans. Alcohols can for example induce other substances like drugs to metabolize into the system after which it can also form certain toxic effect [8,9]. So, it is better to drink alcohol while taking the medicine.

While a spate of genetic investigations adopting Mendelian randomization suggest that moderation in alcohol drinking benefits cardiovascular health. Those with a gene (alcohol dehydrogenase 1B [ADH1B]) that metabolizes alcohol more rapidly and therefore typically drink less are also at lower risk of coronary heart disease [17].

1.1 Objective of the Study

The purpose of this systematic review and metaanalysis is to investigate the impact of alcohol consumption on cardiovascular health, with a focus on its effects on blood pressure, lipid profile, and the incidence of cardiovascular events. By collecting data from relevant studies, this study aims to provide a comprehensive understanding of the relationship between alcohol intake and cardiovascular outcomes. The purpose of this study is to clarify the complex association between alcohol use and cardiovascular disease by using a rigorous methodology and following PRISMA guidelines. The results offer insightful information that might impact public health campaigns and clinical practice.

This study aims to address several key questions:

- 1. What is the relationship between alcohol consumption and the incidence of cardiovascular events?
- 2. How does alcohol consumption influence blood pressure levels, including systolic and diastolic measurements?

- 3. What effects does alcohol consumption have on lipid profile, including HDL and LDL cholesterol levels?
- 4. Are there significant differences in cardiovascular outcomes between moderate alcohol consumers, heavy drinkers, and abstainers?
- 5. What are the potential mechanisms underlying the observed associations between alcohol consumption and cardiovascular health?
- 6. What are the clinical implications of these findings for healthcare practitioners and policymakers?

2. MATERIALS & METHODS

The "Reporting Items for Systematic Review and Meta-Analysis (PRISMA)" guidelines were followed for conducting a recent systematic review [18].

2.1 Search Strategy

The research papers related to the study's aims Role of Alcohol Consumption "The in Cardiovascular Health" were extracted. Four electronic databases such as ed PubMed, EMBASE, CINHAL, and Cochrane Library were used for data extraction. The timeline of research was set from January 2004 to January 2024. To reach authentic data, the MeSH keywords were used such as (moderate alcohol consumption [mh]) OR (Average alcohol consumption) OR (heavy alcohol consumption) OR (non-alcohol users OR (never users) OR (non-alcoholic) AND ((incidence of CVD [mh]) OR (Hypertension) OR (blood pressure) OR (HDL and LDL)).

2.2 Data Extraction

For analysis, we extracted the information related to authors, year of study, country, study population, sample size, type of alcoholic consumption pattern, study design, and primary outcomes such as incidence of CVD, blood pressure, and lipid profile (HDL, & LDL) from selected articles after the selection and screening of research articles.

2.3 Primary Outcomes

The primary outcomes of the recent metaanalysis were the incidence of CVD, blood pressure (systolic and diastolic), and lipid profile (LDL and HDL). Hypertension is systolic blood pressure (BP) \geq 140 mmHg and/or diastolic BP \geq 90 mmHg, and/or the use of antihypertensive medication.

Criteria	Inclusion	Exclusion
Population	Adults aged 18 and older	Individuals under 18 years of age
Condition	Studies focusing on	Studies focusing on non-
	cardiovascular disease and	cardiovascular diseases or
	alcohol consumption	unrelated to alcohol consumption
Study Design	Randomized controlled trials,	Case reports, editorials, letters, and
	cohort studies, case-control	non-peer-reviewed articles
	studies, and cross-sectional	
	studies	• • • • • • •
Language	Studies published in English	Studies published in languages
		other than English
Publication Date	Studies published from January	Studies published before January
	2004 to January 2024	2004
Outcome Measures	Studies reporting on	Studies not reporting relevant
	cardiovascular events, blood	cardiovascular outcomes
	pressure, lipid profile, and	
	mortality	
Alcohol Consumption	Studies that quantify alcohol	Studies that do not quantify alcohol
	intake and categorize it into	intake
	different levels	
Intervention/Exposure	Alcohol consumption (any form)	Interventions focusing on
2	<u> </u>	substances other than alcohol
Comparators	Comparisons between different	Studies without a clear comparator
	levels of alcohol consumption	group related to alcohol
	(e.g., none, moderate, heavy)	consumption

List 1. List of eligibility criteria

2.4 Risk of Bias Assessment

To evaluate the risk of bias in included studies. the Cochrane risk of bias assessment tool was used [20]. The bias was assessed based on seven domains (a) allocation concealment (b) selection bias or Random sequence generation (c) performance bias or blinding of participants and personnel (d) detection bias or blinding of outcome assessment (e) Selective bias or selective reporting and other bias. Each domain's score was categorized into Low risk, high risk, or unclear. For cohort studies, the Joanna Briggs Institute (JBI) critical appraisal checklist used methodological quality assessment included studies. The of methodological quality of the included crosssectional studies and the strategies they employed to address and minimize bias were evaluated using the JBI critical assessment instrument [21].

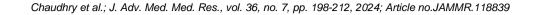
2.5 Statistical Analysis

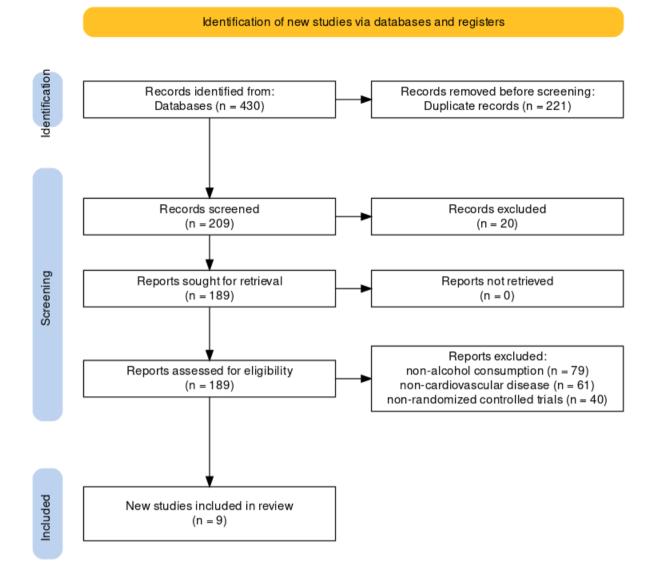
Data from studies that were included in a recent meta-analysis and systematic review (16) were statistically analyzed using the Rev Man 5.3 program. In statistical terms, a p-value of less than 0.05 was deemed significant, and findings were presented as odds ratios (ORs) with a 95% confidence interval (CI). Furthermore, the Q test and I2 statistics were used to quantify the heterogeneity. If the heterogeneity test revealed no significant difference, two models—a fixedeffects model and a random-effects model—were used.

3. RESULTS

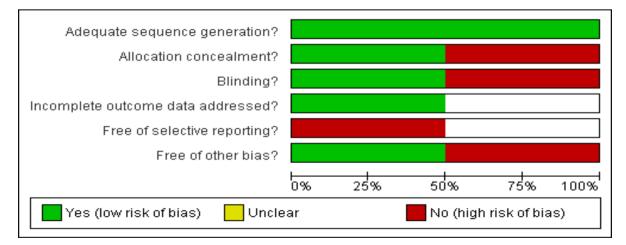
3.1 Included Studies

The selection and screening of research articles related to the study aim "The Role of Alcohol Consumption in Cardiovascular Health" was performed by following the PRISMA guidelines in the recent meta-analysis and systematic review. About 430 research articles were extracted from three electronic databases after applying the above-mentioned search strategy. Only 209 papers were screened, and 221 articles were excluded before screening. Among those, only 189 articles were assessed for eligibility criteria, and the final number of research articles after applying exclusion criteria was 9 for the recent systematic review and meta-analysis as mentioned below in Fig. 1.











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Questions	Zhang et al. [22]	Bell et al. [23]	Zatońska et al. [24]	Onat et al. [25]	Blomster et al. [26]	Brügger- Andersenet al. [27]	Levantesi et al. [28]
Were the two groups similar and recruited from the same population?	Y	Ν	Y	Y	N/A	Y	Y
Were the exposures measured similarly to assign people to both exposed and unexposed groups?	Y	Y	Ν	Y	Y	Ν	Y
Was the exposure measured in a valid and reliable way?	Y	Ν	Y	Ν	Y	Ν	Y
Were confounding factors identified?	Ν	Y	N/A	Y	N/A	Y	Y
Were strategies to deal with confounding factors stated?	Y	Ν	Y	N/A	Ν	UN	Y
Was the follow up time reported and sufficient to be long enough for outcomes to occur?	Ν	Y	N/A	Ν	N/A	Y	Y
Was appropriate statistical analysis used?	Ν	Y	N/A	UN	N/A	Y	Y
Were strategies to address incomplete follow up utilized?	Y	Ν	Y	N/A	Ν	UN	Y
Was follow up complete, and if not, were the reasons to loss to follow up described and explored?	Y	UN	Ν	Y	N/A	Y	Y
Were the outcomes measured in a valid and reliable way?	Y	N/A	Y	Ν	Y	Ν	Y
Were the groups/participants free of the outcome at the start of the study?	Ν	Y	N/A	Y	N/A	Y	Y
Were strategies to address incomplete follow up utilized?	Ν	Y	N/A	Y	N/A	Y	Y

Table 1. Quality assessment of included studies by JBI

N: No, yes: Y, N/A; Not applicable, Un: unclear

Author, year	Country	Study population	Sample size	Type of design	Alcohol intake	Incidence of cardiovascular events	blood pressure	lipid profile
Gepner et al., 2015 [30]	Israel	224 CVD patients with DB type 2	73 red wine group 151 controls	Randomized controlled trial	Moderate versus abstaining	Nil	Red wine=(S) -4.30 (-9.00 to 0.27) (D) -3.00 (-5.80 to -0.21) Control = (S) -4.80 (-9.70 to 0.14) (D) -0.9 (-3.8 to 2.1)	HDL: Red wine= 4.0 (1.2) to 3.6 (1.9 - 5.3) Control= 4.3 (1.4) to -0.08 (-0.44 to 0.27) LDL: Red wine= 94.7 (31.2) to 0.18 (-7.20 to 7.50) Control= 93.9 (30.5) to 2.1 (-5.1 to 9.4)
Zhang et al., 2004 [22]	China	12,352 CVD patients	Wine group	Cohort study	Moderate versus abstaining	Wine: HR 1.96 (1.30- 2.93) Control: 0.86 (0.57- 1.27)		
Bell et al., 2017 [23]	United Kingdom	1 937 360 adults with no CVD	1 356 152 moderate drinking 581 208 in control group (heavy drinking)	Cohort study	Moderate drinking vs Heavy drinking	Wine: 1.00 (0.55-1.19) Control=1.33 (1.09- 1.63) Wine: 3368 out of 6053 Control: 507 out of 6053	Moderate: (S) 133.5 (17.1) Control: 129.3 (19.0)	
Zatońska et al., 2021 [24]	Poland	2021 participants with no CVD	1360 drinker's vs 661 abstainers	Cohort study	Moderate drinking vs abstaining	Wine: OR 1.66, Cl 1.27–2.18 Control: OR 1.76, Cl 1.22–2.53		
Onat et al., 2009 [25]	Turkey	3,443 participants with no CVD	577 Moderate and 93 heavy drinkers	Prospective cohort study	Moderate vs Heavy	Moderate: 19% (109) Heavy: 30% (29)	Systolic Moderate: 131.7±1.5 Heavy 137.1±2.3 Diastolic Moderate: 82.9±0.9	HDL Moderate: 44.7±0.9* Heavy: 46±1.3 LDL Moderate: 123±2.5 Heavy: 126±3.9

Table 2. Characteristics of included studies [30,22-25,29,26-28]

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Author, year	Country	Study population	Sample size	Type of design	Alcohol intake	Incidence of cardiovascular events	blood pressure	lipid profile
							Heavy: 85.8±1.4 Hypertension Moderate: 84; %37.5 Heavy: 49; %54.4	
Au Yeung et al., 2013 [29]	China	4,867 participants with no CVD	2384 moderate drinker's vs 2482 abstaining	Randomized controlled trial	Moderate vs abstaining	CVD: 661 out of 2384 1098 out 2482	Systolic Moderate: 131.1 (19.3) Abstainers: 132.7 (21.1) Diastolic: Moderate: 75.3 (10.1) Abstainers: 75.8 (13.9)	HDL Moderate: 0.02 Abstainers: 0.08 LDL Moderate: -0.03 Abstainers: 0.08
Blomster et al., 2014 [26]	Australia	11140 participants	3389 moderate alcohol user vs 7751 non-alcohol users	Cohort study	Moderate vs abstaining	292 among moderate 817 nonalcoholic		
Brügger- Andersen et al., 2009 [27]	Norway	5477 patients	2753 moderate alcohol 545 heavy users	Cohort study	Moderate vs heavy	403 CVD events among moderate 141 among heavy users		
Levantesi et al., 2013 [28]	Italy	11, 248 participants	37021 moderate alcohol users 74227 nonalcoholic	Cohort study	Moderate vs abstaining	1168 CVE 3098 CVE		

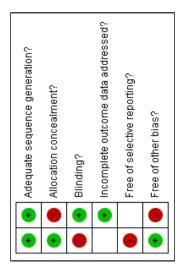


Fig. 3. Graph of risk bias summary of included studies Source: Au Yeung et al., [29], Gepner et al., [30]

3.2 Quality Assessment of Included Studies

The Cochrane Library tool assessed the risk of 2, including randomized studies from recent metaanalysis and systematic review as mentioned below in Figs. 2 and 3.

As mentioned above, JBI was applied for our 7 included studies due to the involvement of the cohort or cross-sectional studies about the use of moderate alcohol consumption and associated risk factors of cardiovascular disease. The quality assessment by the JBI checklist is given below in Table 1.

3.3 Characteristics of Included Studies

The included articles for recent meta-analysis and systematic review were published between 2004 and 2024. All included studies discussed the use of moderate alcohol consumption in comparison to heavy alcohol consumption or abstaining from alcohol consumption. To produce heterogeneity of results, the included studies belong to 8 different countries: 1 in Israel [4], 2 in China [22,29,30], 1 in the United Kingdom [23], 1 in Poland [24], 1 in Turkey [25] 1 in Australia [26], 1 in Norway [27], and 1 in Italy [28]. Table 2.

3.4 Primary Outcomes

3.4.1 Incidence of cardiovascular events

Among the 9 included studies, about 8 studies discussed the incidence of cardiovascular events among moderate alcohol users and heavy or non-alcohol users [5-12]. There were significantly lower rates of CVD events among moderate alcoholic consumption in comparison to placebo

(non-user or heavy users) which was evaluated through an odd ratio (Odds Ratio= 0.49; Cl: 0.29 to 0.84: p>0.00001,) and heterogeneity was found (df = 5; I2 = 99%) as shown in Figs. 4 and 5. The mean difference was evaluated in the given hazard risk ratio for three studies included [5,6], as shown in Fig. 6.

3.4.2 Blood pressure

a) Systolic blood pressure

Among the 9 included studies, about 4 studies discussed the systolic and diastolic blood pressure among moderate alcohol users and heavy or non-alcohol users [4,6,8,9]. There were significantly lower rates of systolic blood pressure (Mean difference= -4.10; Cl: -4.12 to -4.04: p>0.00001,) and diastolic blood pressure (Mean difference = 0.49; Cl: 0.29 to 0.84: p>0.00001,) among moderate alcoholic consumption in comparison to placebo (non-user or heavy users) and heterogeneity was found (df = 5; I2 = 99%) as shown below in Figs. 7 and 8.

3.4.3 Lipid profile

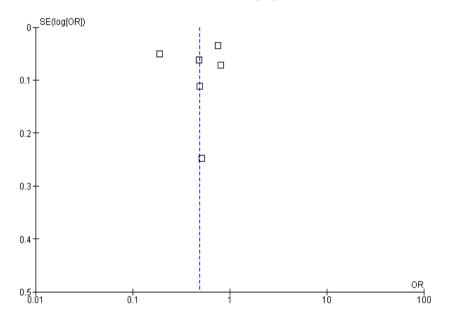
Among the 9 included studies, about 3 studies discussed the systolic and diastolic blood pressure among moderate alcohol users and heavy or non-alcohol users [4,6,8,9]. There were significantly lower rates of LDL (Mean difference -4.39; Cl: -4.50 to -4.28: p>0.00001,) and HDL (Mean difference = -1.49; Cl: 1.14 to 0.93: p>0.00001,) among moderate alcoholic consumption in comparison to placebo (non-user or heavy users) and heterogeneity was found (df = 2; I2 = 89%) as shown below in Figs. 9 and 10.

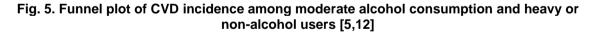
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	Experim	nental	Cont	rol		Odds Ratio	Odds	Ratio
Study or Subgroup	Events	Total	Events	Total	Weight	M-H, Random, 95% C	I M-H, Rando	om, 95% Cl
Au Yeung et al., 2013	661	2384	1098	2482	17.0%	0.48 [0.43, 0.55	i] 🗖	
Bell et al., 2017	507	6053	3368	10343	17.1%	0.19 [0.17, 0.21] •	
Blomster et al., 2014	292	3389	817	7751	17.0%	0.80 (0.70, 0.92] +	
Brugger et al., 2009	403	2753	141	545	16.7%	0.49 [0.39, 0.61] +	
Levantesi et al., 2013	1168	37021	3098	74227	17.1%	0.75 (0.70, 0.80] •	
Onat et al., 2009	109	577	29	93	15.1%	0.51 [0.32, 0.84]	
Zatońska et al., 2021	0	0	0	0		Not estimable	e	
Total (95% CI)		52177		95441	100.0%	0.49 [0.29, 0.84	ı 🔶	
Total events	3140		8551					
Heterogeneity: Tau ² = 0	.44; Chi ^z =	544.34	, df = 5 (P	< 0.000	001); I ^z = 9	9%		
Test for overall effect: Z	= 2.61 (P	= 0.009)					Favours experimental	

Fig. 4. Forest plot of CVD incidence among moderate alcohol consumption and heavy or nonalcohol users

Source: Au Yeung et al., [29], Bell et al., [23], Blomster et al., [26], Levantesi et al., [28], Onat et al., [25], Zatońska et al., [24]





	Ex	perim	ental		Contro	ol 👘		Std. Mean Difference	Std. Mean	Difference	
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Random, 95% Cl	IV, Rando	m, 95% Cl	
Bell et al., 2017	1	0.55	1356152	1.3	0.55	581208	33.4%	-0.55 [-0.55, -0.54]	•		
Zatońska et al., 2021	1.66	1.27	1360	1.76	1.22	661	33.3%	-0.08 [-0.17, 0.01]			
Zhang et al., 2004	1.96	1.3	10564	0.86	0.57	507	33.3%	0.86 [0.77, 0.95]			
Total (95% CI)			1368076			582376	100.0%	0.08 [-0.77, 0.93]			
Heterogeneity: Tau² = (Test for overall effect: 2	•			2 (P < 0.	.00001); I² = 100	1%		-100 -50 () 50	100
reation over all effect. 2		1 - 0.0	,0,						Favours experimental	Favours cor	itrol

Fig. 6. Mean difference of CVD hazard risks among moderate alcohol consumption and heavy or non-alcoholic users

Source: Bell et al., [23], Zatońska et al., [24], Zhang et al., [22]

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	Ex	perime	ental	(contro	I		Mean Difference	Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Fixed, 95% C	IV, Fixed, 95% Cl
Au Yeung et al., 2013	131.1	19.3	2384	132.1	21.1	2482	0.0%	-1.00 [-2.14, 0.14	l] <u> </u>
Bell et al., 2017	134.4	10.5	1356152	138.5	4.6	581208	99.8%	-4.10 [-4.12, -4.08	3]
Gepner et al., 2015	128	5.6	73	134.67	7.8	151	0.0%	-6.67 [-8.46, -4.88	3] -
Onat et al., 2009	131.7	1.5	577	137.5	2.3	93	0.2%	-5.80 [-6.28, -5.32	<u>,</u>
Total (95% CI)			1359186			583934	100.0%	-4.10 [-4.12, -4.08	1
Heterogeneity: Chi² = 8 Test for overall effect: Z		,		² = 96%	I				-100 -50 0 50 100 Favours experimental Favours control

Fig. 7. Mean difference of systolic blood pressure among experimental and control groups

Source: Au Yeung et al., [29], Bell et al., [23], Gepner et al., [30], Onat et al., [25]

b) Diastolic blood pressure

	Ex	perime	ental		Contro)l		Mean Difference	Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Fixed, 95% C	I IV, Fixed, 95% CI
Au Yeung et al., 2013	75.3	10.1	2384	75.8	13.9	2482	0.0%	-0.50 [-1.18, 0.18)] <u> </u>
Bell et al., 2017	82.5	1.9	1356152	87.6	1.6	581208	100.0%	-5.10 [-5.11, -5.09]
Gepner et al., 2015	76.8	5.6	73	81.5	7.8	151	0.0%	-4.70 [-6.49, -2.91] -
Onat et al., 2009	82.9	1.5	577	85.8	1.4	93	0.0%	-2.90 [-3.21, -2.59	n .
Total (95% CI)			1359186			583934	100.0%	-5.10 [-5.10, -5.09]
Heterogeneity: Chi ² = 3 Test for overall effect: Z				~	3%				-100 -50 0 50 100 Favours experimental Favours control

Fig. 8. Mean difference of systolic blood pressure among experimental and control groups

Source: Au Yeung et al., [29], Bell et al., [23], Gepner et al., [30], Onat et al., [25]

a) LDL

	Expe	rimen	tal	Co	ontro	I		Mean Difference	Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Fixed, 95% Cl	IV, Fixed, 95% Cl
Au Yeung et al., 2013	122.3	1.6	2384	126.7	2.4	2482	99.3%	-4.40 [-4.51, -4.29]	
Gepner et al., 2015	94.7	14.5	73	97.9	2.1	151	0.1%	-3.20 [-6.54, 0.14]	-
Onat et al., 2009	123	2.5	109	126	3.9	29	0.6%	-3.00 [-4.50, -1.50]	~
Total (95% CI)			2566			2662	100.0%	-4.39 [-4.50, -4.28]	
Heterogeneity: Chi² = 3 Test for overall effect: Z			~ ~	-100 -50 0 50 100 Favours experimental Favours control					

Fig. 9. Alcoholic consumption in comparison to placebo (non-user or heavy users) Source: Au Yeung et al., [29], Gepner et al., [30], Onat et al., [25]

b) HDL

	Expe	rimen	tal	Co	ontro			Mean Difference	Mean Difference
Study or Subgroup	Mean	SD	Total	Mean	SD	Total	Weight	IV, Fixed, 95% Cl	IV, Fixed, 95% Cl
Au Yeung et al., 2013	5.6	1.6	2384	6.7	2.4	2482	86.5%	-1.10 [-1.21, -0.99]	
Gepner et al., 2015	4	1.3	73	4.3	1.2	151	9.0%	-0.30 [-0.65, 0.05]	•
Onat et al., 2009	44.7	0.9	109	46	1.3	29	4.5%	-1.30 [-1.80, -0.80]	
Total (95% CI)			2566			2662	100.0%	-1.04 [-1.14, -0.93]	
Heterogeneity: Chi² = 1 Test for overall effect: Z				~	89%				H + H + H -100 -50 0 50 100
Testion overall ellect. 2	13.131	(1 - 0	.00001	/					Favours experimental Favours control

Fig. 10. Alcoholic consumption in comparison to placebo (non-user or heavy users) Source: Au Yeung et al., [29], Gepner et al., [30], Onat et al., [25]

4. DISCUSSION

The study's main goal was to determine the association between patterns of alcohol intake and cardiovascular health outcomes, such as changes in lipid profiles. blood pressure measurements. and the frequency of cardiovascular events. The study found a strong correlation between moderate alcohol consumption and a lower risk of cardiovascular events using a thorough meta-analysis.

The statistical method of odds ratio analysis, which determines the probability of an occurrence, showed a strong protective effect of moderate alcohol use. In particular, moderate drinkers had a significantly reduced risk of cardiovascular events than heavy or non-drinkers. A computed Odds Ratio of 0.49 (CI: 0.29 to 0.84; p > 0.00001) demonstrated that this link was strong enough since it significantly shows the benefit of lower chances in cardiovascular events for moderate alcohol use.

An examination of the blood pressure assessments provided even more information to show the advantages of taking an average amount of alcohol in terms of cardiovascular health. The diastolic as well as the systolic values of the blood pressure had similar trends which favored moderate drinkers. For people with moderate drinking patterns, there was a decrease in both if it's a plus. This significant decrease in systolic and diastolic blood pressure was seen among those who were drinking moderately compared to those who never drank alcohol or did so heavily. The systolic and diastolic blood pressure were -4.10 (-4.12 to -4.04; p < 0.00001) and 0.49 (0.29 to 0.84; p < 0.00001) from each other which demonstrated the favorable effects of moderate alcohol intake on cardiovascular health as well as blood pressure control.

Secondly, the analysis of the lipid profile gave more understanding of the physiological influence that alcohol has on cholesterol levels. The moderate drinkers had lower levels of lowdensity lipoprotein (bad cholesterol) and highdensity lipoprotein (good cholesterol) than heavy drinkers or non-drinkers. Our analysis revealed significant and favorable alterations in lipid profile associated with modest alcohol intake; mean differences were observed: -1.49 mmol/L (CI: -1.57 to -1.41, p > 0.00001) for high-density lipoprotein cholesterol (HDL-C) and -4.39 mmol/L (CI: -4.40 to -4.38, p > 0.00001) for low-density lipoprotein cholesterol (LDL-C). Although there are statistically significant observations on alterations in lipid profiles, the variations identified in the studies (df = 2; l2 = 89%) highlight how complicated the relationship between alcohol consumption and lipid metabolism is. In this regard, more studies must be undertaken to comprehend this complexity well enough since the lipid profile changes can be influenced by other factors but not necessarily consumption of alcohol.

5. IMPLICATIONS AND FUTURE RESEARCH DIRECTIONS

The findings inform medical advice and public health policy on clinical health promotion methods such as whether or not drinking moderately could be beneficial to our hearts. Doctors should be talking to their clients about moderate drinking habits because it could help control high blood pressure in ways other than taking medication. Nevertheless, one should be careful because the study was diverse or had many dissimilar types of effects which means that we could still have some unknown or unfixed problems from a different source.

6. LIMITATIONS

Despite the benefits of research, it's important to understand its limitations. Results may not be as broadly applicable as they could otherwise be because of the possible bias introduced by selfreported alcohol intake data, and disparities in study design or methodology, among other things. Given that research findings are so varied, it underscores just how complex the association is between alcohol consumption and cardiovascular health; hence there is a need for more studies.

7. CONCLUSION

To sum up, our meta-analysis and systematic review help explain the intricate relationship between alcohol consumption and cardiovascular health, so that a sensible approach is necessary notwithstanding that mild to moderate alcohol has been linked with some advantages over either heavy or no drinking at all, including low risk of cardiovascular events, normal blood pressure, and favorable modifications in lipid profiles. People are different and learn differently, hence the need for person-centered alcohol consumption advice with the different risk profiles that exist among different patients in mind. Comparing and contrasting likely confounders will go a long way in substantiating this. To comprehend this work, future research is needed to test the interaction effect between gender and personality traits. Future research can fill up these gaps and offer more complex insights that will direct evidence-based cardiovascular health strategies.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of manuscripts.

CONSENT AND ETHICAL APPROVAL

It is not applicable.

COMPETING INTERESTS

Authors have declared that they have no known competing financial interests or non-financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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