

ROLE OF ALCOHOL IN DEVELOPMENT OF CHD (CORONARY HEART DISEASE)Vijna¹, Amita Sinha²¹Tutor, Department of Community Medicine, Nalanda Medical College and Hospital, Patna, Bihar, India.²Associate Professor, Department of Community Medicine, Nalanda Medical College and Hospital, Patna, Bihar, India.Received : 05/01/2023
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2023; 5 (5); 493-496**Abstract**

Background: Epidemiological studies of middle-aged populations generally find the relationship between alcohol intake and the risk of coronary heart disease (CHD) and stroke to be either U- or J-shaped. This review describes the extent that these relationships are likely to be causal, and the extent that they may be due to specific methodological weaknesses in epidemiological studies. The consistency in the vascular benefit associated with moderate drinking (compared with non-drinking) observed across different studies, together with the existence of credible biological pathways, strongly suggests that at least some of this benefit is real. However, because of biases introduced by: choice of reference categories; reverse causality bias; variations in alcohol intake over time; and confounding, some of it is likely to be an artefact. For heavy drinking, different study biases have the potential to act in opposing directions, and as such, the true effects of heavy drinking on vascular risk are uncertain. However, because of the known harmful effects of heavy drinking on non-vascular mortality, the problem is an academic one. Studies of the effects of alcohol consumption on health outcomes should recognise the methodological biases they are likely to face, and design, analyse and interpret their studies accordingly. While regular moderate alcohol consumption during middle-age probably does reduce vascular risk, care should be taken when making general recommendations about safe levels of alcohol intake. In particular, it is likely that any promotion of alcohol for health reasons would do substantially more harm than good.

INTRODUCTION

Medical and political discussions of the health effects of alcohol should give prominence to the individual and social damage caused by alcohol.^[1] In the interests of public health, it is right that any discussion should begin and end with these problems. In the middle, however, perhaps a small place may be reserved for continued exploration of why moderate drinkers appear to have a lower mortality risk, and particularly a lower incidence of coronary heart disease (CHD).

As suggested recently, non-drinkers may include a number of ex-drinkers who gave up because of ill-health.^[2] Hence a high mortality would not be surprising. This is plausible, but other evidence suggests different reasons for the CHD advantage of moderate drinkers.

A major problem concerns the varying definitions of 'heavy' and 'moderate'. There is agreement that daily

consumption of more than 80 g of ethanol is 'heavy'.^[3] This is the amount of alcohol contained in five pints of beer, or a bottle of table wine, or one third of a bottle of spirits (Table 1). However, others would put the dividing line between moderate and heavy at a lower level than this.^[4] Presumably an appropriate way to define 'heavy' is the level above which alcohol-associated problems emerge; but this is a complex subject since alcohol is associated with a wide range of medical and social problems. The question considered here relates only to coronary heart disease.

It is accepted that heavy alcohol consumption can have a direct toxic effect on the myocardium.^[5]

Aim and Objective

The data on two questions are reviewed: does heavy alcohol increase the risk of coronary heart disease (CHD)? And moderate intake protective?

MATERIALS AND METHODS

It was the experimental studies with random controlled trial. The duration of the study is November 2021 October 2022.

Does Heavy Alcohol Consumption Increase CHD Risk

Heavy alcohol consumption is related to increased total mortality, but the evidence for a relationship with cardiovascular mortality or morbidity is not consistent.

Regional Comparison

In the Regional Heart Study of 3 Prime Areas of Patna in Bihar Shaper and colleagues show a positive correlation between the proportion of heavy drinkers in a town and mortality from CHD.^[6] These data do not relate to individuals; they examine one group characteristic heavy drinking and relate it to another CHD mortality.

Studies Of Alcoholics or Problem Drinkers

Two studies in industry of non-institutionalized men whose drinking interfered with their work, in the Du Pont company⁸ and in Chicago,^[9] showed these problem drinkers to have an increased risk of dying from cardiovascular disease. The relative risk was 2.3 for CHD in Du Pont, and 4.0 in Chicago. In Chicago, making adjustments for age, smoking and other risk factors reduced the mortality ratio a little, but it was still elevated.

In Sweden, Wilhemsen et al. took registration with the Swedish Temperance Board as an indicator of heavy alcohol consumption and found an increased rate of non-fatal CHD and of sudden cardiac death, independent of blood pressure and smoking.^[10]

Other studies have concentrated not on problem drinkers or alcoholics but on actual alcohol consumption; almost all have shown heavy drinkers to have either a lower or the same risk of CHD as non-drinkers. Apparently the only exceptions are one study from Chicago that showed a non-significant excess mortality,^[11] and a twin study that showed an excess of angina pectoris.^[12]

How Might Alcohol Protect Against CHD

Type of Alcohol

If one type of alcohol beverage were more strongly 'protective' against CHD, this would make it more likely that it was not alcohol per se. The findings on this point do not, at the moment, implicate one type of drink over another. St Leger et al.^[13] found the inverse association between countries to be strongest with wine, but Laporte et al. found the inverse association with time trends in the USA to be strongest with beer.^[15] In three studies,^[18,7] different types of alcoholic drinks were all shown to be more or less equally associated with lower CHD risk. The other studies did not distinguish type of alcoholic drink. Nevertheless, it remains a possibility that components of alcoholic drink other than ethanol are responsible for a 'protective' effect.

Possible Mechanisms

Atheroma

There is not general agreement, but there have been reports of less atheroma in alcoholics at autopsy.^[15] In general these were studies of heavy, not moderate drinkers.

One study of patients undergoing coronary angiography,^[20] found significantly lower occlusive scores in moderate than in non-drinkers. Such studies are difficult to interpret because of the biased selection of patients.

Lipids

Several studies have shown HDL cholesterol levels to be higher in moderate drinkers,^[15,19] and high levels of HDL cholesterol are associated with lower CHD risk. However, the fraction associated with lower CHD risk is HDL₂, whereas alcohol may increase the HDL₃ fraction, although this has not been definitely established.

Thrombocytopenia

Alcohol in large amounts can produce thrombocytopenia and decreased platelet aggregation. Meade has reported that drinkers have lower fibrinogen levels and higher fibrinolytic activity than non-drinkers,^[20] these effects could protect against CHD.

Is The Negative Association Casual

There is some evidence of an increased risk of CHD in heavy drinkers. This is not a crucial public health question, however, as there is sufficient evidence of the hazards of heavy drinking to make it undesirable, regardless of a possible relation with CHD.

The evidence that moderate alcohol consumption may be protective may be assessed in relation to the formal criteria for a causal association.

Strength

The relative risk for moderated alcohol consumption is of the order of 0.5. It is quite conceivable that some third factor(s) may account for an observed association of this order of strength.

Dose Response

An inverse dose-response relationship has not been found consistently, possibly due to inaccuracies in determining alcohol consumption. Whatever the reason, this is a weakness in the current evidence.

Temporal Sequence

A number of studies have established that non-drinking preceded the onset of CHD.

Consistency

One of the strongest arguments in favour of causality is that the inverse association with alcohol has been found in several different populations, in case-control and longitudinal studies, in international comparisons and in analyses of mortality time trends. Each of these types of study has its own weaknesses. Consistent findings from such varied sources make it more likely that moderate alcohol consumption is protective.

Independence

Where studied, the association between non-drinking and CHD has been found to be independent of other major cardiac risk factors.

Plausibility

The effect of alcohol on HDL cholesterol offers a plausible mechanism (or did so, until recent doubts arose on the relevance of the HDL fraction influenced by alcohol), as does the effect on haemostasis.

Specificity

There is some evidence that deaths from other causes may be commoner in non-drinkers, but not to the same extent as cardiovascular disease, 24 and this has been found less consistently.

In summary, the evidence is far from complete; but it does point towards a protective effect of moderate alcohol consumption. If there is a level of alcohol which is no longer 'safe' for CHD, it is probably in excess of six drinks per day (approx 70 g alcohol). If the apparent protective effect is due to confounding variables, they have yet to be identified.

What Recommendations Should Be Made?

If, as an interim judgement, we assume that the protective effect of moderate alcohol consumption is likely to be causal, two further aspects must be considered in making recommendations: what is the upper limit of 'moderate' and what are the likely effects of recommending moderate alcohol intake?

The figure from the Whitehall study shows a U-shaped relationship of mortality to alcohol; we took

34 g alcohol per day (about four drinks) as the start of our 'heavy' category of consumption. It is difficult to know at precisely what level of alcohol consumption the non-CVD mortality starts to increase, but data from other studies show blood pressure to be higher with four drinks (or even less) per day.

The Royal College of Psychiatrists has recommended²² a maximum limit of twice this amount (about eight drinks per day), but this seems to be too high. There is an association between the mean level of alcohol consumption of a community and the proportion of problem drinkers,^[14] and anything that encourages an increase in average consumption is likely to lead to disastrous consequences in a minority, as well as to more widespread social costs and an increase in road accidents. These considerations, linked with the fact that the role of moderate intake in protecting against CHD is not certain, lead the author to agree with the conclusion of the recent WHO Expert Committee on the Prevention of CHD: 'Increased alcohol intake is not recommended as a preventive measure in CHD, either in populations or in individuals.'

Table 1: Case-control studies of alcohol and coronary heart disease (CHD)

Study population	Sex	amount	none	Moderate	Heavy	Controlled for risk factors.
Non-fatal MI v. hospital controls	M&F	6 drinks/day	2.0	1.0	0.5(0.2-0.1 ^b)	Yes
Fatal CHD v. population controls	M	1/week to 1/month	2.25 ^c	1.5	1.0	Yes
Fatal cases v. neighbourhood controls	M	2 oz (59 ml) alcohol/day	2.0	0.1-0.3 ^a	2.0	Yes
Participants in pre-paid health plan	M&F	3-5 drinks/day				Yes
Hospitalized MI v. matched controls		--	1.0	0.5	0.4 ^d	
Sudden cardiac death v. matched controls		--	1.0	0.6	0.6	
Fatal CHD v. living controls (same community)	F	2 drinks/day	1.0	0.3 ^e		

P < 0.001

b95% confidence intervals. cNot significantly different.

dP < 0.05 (heavy drinkers v. all others), P < 0.01 (non-drinkers v. all others).

eP < 0.01.

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