Alcohol intake and mortality in middle aged men with diagnosed coronary heart disease

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Abstract

Objective—To examine the effects of alcohol on risk of mortality from coronary heart disease (CHD), cardiovascular disease, and all causes in men with established CHD.

Methods and results—In a population based prospective study of 7169 men aged 45–64 years followed for a mean of 12.8 years, 655 men (9.1%) had a physician diagnosis of CHD (myocardial infarction 455, angina only 200). In these 655 men, there were 294 deaths from all causes including 175 CHD deaths. Ex-drinkers had the highest risk of CHD, cardiovascular mortality, and all cause mortality even after adjustment for lifestyle characteristics and pre-existing disease. Using occasional drinkers as the reference group, lifelong teetotallers, occasional drinkers, and light drinkers all showed similar risks of mortality from CHD, cardiovascular disease, and all causes. Moderate/heavy drinkers showed increased risk of mortality from CHD, cardiovascular disease, and all causes compared to occasional drinkers. The adverse effect of moderate/heavy drinking was confined to the 455 men with previous myocardial infarction (adjusted relative risk for all cause mortality 1.50, 95% confidence interval 1.01 to 2.23). In contrast to lighter drink-ing, giving up smoking within five years of the start of follow up was associated with a consider-able reduction in risk of all cause and cardiovascular mortality compared to those who continued to smoke.

Conclusion—Compared to occasional drinking, regular light alcohol consumption (1–14 units per week) in men with established coronary heart disease is not associated with any significant benefit or deleterious effect for CHD, cardiovascular disease or all cause mortality. Higher levels of intake (\geq 3 drinks per day) are associated with increased mortality in men with previous myocardial infarction. In contrast, smoking cessation in men with established CHD substantially reduces the risk of mortality.

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Keywords: coronary heart disease; alcohol consumption; mortality risk; smoking cessation

It is well established that light to moderate drinking is associated with lower risk of major coronary heart disease (CHD) events.¹⁻¹² In the majority of prospective studies relating alcohol intake to cardiovascular morbidity and all cause mortality, men with diagnosed CHD have been excluded or have been included in the large group of subjects with pre-existing disease (variously defined).³⁻¹⁶ Most studies which have examined the effects in men with and without pre-existing cardiovascular diseases have found the "protective" effect of light to moderate alcohol intake in both groups of men.9-15 The role of alcohol specifically in men with established CHD who are at greater increased risk of CHD mortality is therefore less certain. While clinical studies have suggested that alcohol drinking may be harmful in men with established CHD,¹⁷ evidence from population prospective studies to support this is lacking; given the consistent evidence for the beneficial effects of light to moderate alcohol intake in population studies it may even be assumed that alcohol intake plays a beneficial role in secondary prevention of CHD. Recently the US physicians' health study reported that a light to moderate amount of alcohol in men with previous myocardial infarction is associated with "a slight but clinically important decrease in total mortality compared with those who (usually) never or rarely drank alcohol".18 Lifelong teetotallers and exdrinkers were not separated. In this predominantly lightly drinking population of American physicians, the effects of moderate or heavy drinking (\geq 3 drinks per day) could not be assessed.

Our study examines the relation between alcohol intake and risk of CHD and all cause mortality in 655 men with a history of diagnosis of CHD (myocardial infarction or angina, or both) drawn from the general population, separating lifelong teetotallers and ex-drinkers. It also compares the effects of alcohol intake and cigarette smoking cessation on CHD and all cause mortality in these men as a contrast in effect.

Subjects and methods

The British regional heart study is a prospective study of cardiovascular disease involving 7735 men aged 40–59 years selected from the age–sex registers of a single group general practice in each of 24 towns in England, Wales, and Scotland and examined from January 1978 to July 1980. Men with pre-existing cardiovascular disease or receiving regular medical treatment were not excluded and the overall response rate was 78%. The criteria for selecting the town, the general practice, and the subjects as well as the methods of data collection have been reported previously.¹⁹ Research nurses administered to each man a standard questionnaire (Q1), which included questions

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on smoking habits, alcohol intake, and medical history. Several physical measurements were made and blood samples (non-fasting) were taken for measurement of biochemical and haematological variables. Five years after the initial examination (1983 to 1985), a postal questionnaire (Q5) similar to the one administered at screening was sent to all surviving men; detailed information on medical history, and changes in smoking and drinking behaviour, and in other risk factors, was obtained from 98% (n = 7275).

PRE-EXISTING DISEASE

At both the initial screening (Q1) and five years later (Q5) men were asked whether a doctor had ever told them that they had angina or a myocardial infarction (heart attack, coronary thrombosis), stroke, diabetes, and a number of other disorders. They were also asked for details of any regular medication.

ALCOHOL INTAKE

Alcohol consumption was recorded at initial screening (Q1) using questions on frequency, quantity, and type, similar to those used in the 1978 general household survey.²⁰ The men were questioned about frequency and quantity of alcohol intake, resulting in eight drinking categories: non-drinkers, occasional drinkers (special occasions or 1-2 drinks per month), weekend drinkers (1-2, 3-6 or > 6 drinks per)day), and men drinking daily or on most days (1-2, 3-6 or > 6 drinks per day). These categories were the only choices provided; the "> 6 drinks per day" is an open ended category. One UK unit of alcohol (one drink) is defined as half a pint of beer, a single measure of spirits, or a glass of wine (approximately 8-10 g alcohol). Heavy drinking refers to those drinking > 6 drinks per day either daily or on most days in the week (> 42 drinks per week). Twenty five biochemical and haematological measurements on a single blood sample taken at the time the questionnaire was completed by the men in this study indicated that the reported levels of alcohol consumption were valid on a group basis.²¹ Five years later (Q5), the men were asked about their past drinking habits in addition to questions on their current alcohol consumption. Those who said they were non-drinkers at Q5 were asked whether they had been drinkers in the past and, if so, what their past alcohol consumption had been. No biochemical/haematological validation was carried out on this occasion. Complete information on alcohol consumption at both Q1 and Q5 was obtained from 7169 men. The men were classified on the basis of their estimated reported weekly intake⁸ at O5:

- Lifelong teetotaller—non-drinkers at Q5 who did not report drinking in the past and were non-drinkers or occasional drinkers at Q1.
- (2) Ex-drinkers—non-drinkers at Q5 who at Q5 reported previous regular drinking, and non-drinkers at Q5 who were regular drinkers at Q1.
- (3) Occasional (< 1 per week)—those who reported occasional drinking at Q5.

- (4) Light (1-15 units per week)—weekend 1-2, 3-6, and daily 1-2 at Q5.
- (5) Moderate (16-42 units per week)—daily 3-6 and weekend > 6 at Q5.
- (6) Heavy (> 42 units per week)—daily > 6 drinks per day at Q5.

Lifelong teetotallers and ex-drinkers are separated because we have shown that they have different cardiovascular and total mortality outcomes, while regular drinkers-light, moderate, and heavy-have similar total mortality outcomes.8 Between Q1 and Q5, occasional drinkers tend to remain stable, while heavy and moderate drinkers tend to decrease their intake.²² Occasional drinkers are used as the reference group in this study, as the non-drinking groups have been shown to be an inappropriate baseline group for studying the effects of alcohol on health and disease8 23; also, occasional drinkers constitute a large group and proved to be the most stable of the drinking categories over the five year period preceding follow up.²⁴

LIFESTYLE CHARACTERISTICS Smoking

From the combined information at screening and five years later the men were classified as those who had never smoked, ex-smokers at both Q1 and Q5, ex-smokers at Q5 only, and two groups of current cigarette smokers at Q5 (1-19 and > 20 per day).

Social class

The longest held occupation of each man was recorded at screening and the men were grouped into one of six social classes: I, II, III non-manual, and III manual, IV, and V. Those whose longest occupation was in the Armed Forces formed a separate group.

Body mass index

At Q5 the men were asked to state their weight; body mass index (BMI, weight/height²) was then calculated (kg/m²) for each man based on their reported weight and on measured height at initial screening.

FOLLOW UP

All men, whether or not they had evidence of CHD at initial examination, were followed up for all cause mortality and for cardiovascular morbidity from the initial screening in 1978-80.25 All deaths occurring in the period up to December 1996 have been recorded and follow up has been achieved for 99% of the cohort. However, this report is concerned only with the men who completed the fifth year questionnaire; thus mortality follow up since the fifth year questionnaire is presented-a mean follow up period of 12.8 years (range 11.5 to 14.0 years). Information on death was collected through the established "tagging" procedures provided by the National Health Service registers in Southport (England and Wales) and Edinburgh (Scotland). Fatal CHD events included all deaths with CHD as the underlying cause (International Classification of Disease, ninth revision, codes 410-414). All death certificates relating to cardiovascular

disease were explored in detail when it appeared that coding to cardiovascular disease was not appropriate or if cardiovascular disease was not the attributed code when it might have been. In such cases, the findings were investigated by correspondence with the certifying doctor and the hospital concerned.

MEN WITH PHYSICIAN DIAGNOSED CHD

This group comprised men with a recall of a physician diagnosis of CHD (heart attack or angina) at Q1 or Q5, and those who had suffered a major non-fatal myocardial infarction event before Q5 based on biennial reviews of each patient's general practice records, including all hospital reports and correspondence through to the end of the study, supplemented by personal questionnaires at the fifth (Q5) and 12th (Q92) years after screening. Of the 7169 men who had complete data on alcohol history at Q1 and Q5, 655 men had a history of CHD. In this cohort, validation of patient recall of a doctor diagnosis of CHD (angina or heart attack) has been established by comparison of recall with the patient's records.^{26 27} This report is primarily concerned with these 655 men from the study.

STATISTICAL METHODS

The Cox proportional hazards model was used to assess the independent contributions of alcohol intake to the risk of mortality and major CHD events and to obtain the relative risks adjusted for age and the other risk factors.²⁸ In the adjustment, age and body mass index were fitted as continuous variables and smoking (five groups), social class (seven groups), and recall of diabetes (yes/no) were fitted as categorical variables. Direct standardisation was used to obtain age adjusted rates per 1000 person years, using the study population as the standard.

Results

During the mean follow up period of 12.8 years from the fifth year questionnaire in the 655 men with history of CHD, there were 294 deaths from all causes (46.1/1000 person years), of which 208 were attributed to cardiovascular causes (32.7/1000 person years) mainly caused by CHD (175 deaths; 27.5/1000 person years).

ALCOHOL AND ALL CAUSE MORTALITY

The 655 men were divided at Q5 into six categories on the basis of their average weekly estimate: lifelong teetotallers (n = 43), exdrinkers (n = 59), occasional drinkers (n = 199), light drinkers (n = 230), moderate drinkers (n = 104), and heavy drinkers (n=20). Because of the small number of men in the heavy drinking group these men were combined with the moderate drinkers and five groups were used. There were only 86 deaths from non-cardiovascular causes. Table 1 shows the age adjusted rates and relative risks for CHD, cardiovascular, non-cardiovascular, and all cause mortality, and the relative risks adjusted in addition for lifestyle characteristics-that is, smoking, social class, BMI, and pre-existing disease (diabetes, stroke, and regular medication). Ex-drinkers had by far the highest rates of mortality from CHD, cardiovascular, non-cardiovascular, and all causes even after adjustment for lifestyle characteristics, although this was only significant for all cause mortality, probably because of the relatively small numbers involved. There was little difference in risk of CHD, cardiovascular, non-cardiovascular, and all cause mortality between lifelong teetotallers, occasional drinkers, and light drinkers. Moderate/heavy drinkers showed increased risk of CHD, cardiovascular mortality, and all cause mortality

Table 1 Mortality from CHD, cardiovascular, non-cardiovascular, and all causes in 655 men with established CHD at Q5

	Number of men	Number of cases	Rates*	Relative risk (95% CI)†		
Alcohol intake (Q5)				А	В	
CHD mortality (n=175)						
Teetotallers	43	12	27.6	1.04 (0.55 to 1.95)	1.14 (0.60 to 2.18)	
Ex-drinkers	59	21	40.8	1.53 (0.92 to 2.55)	1.47 (0.88 to 2.48)	
Occasionals	199	52	26.6	1.00	1.00	
Light	230	52	22.5	0.85 (0.58 to 1.24)	0.93 (0.63 to 1.37)	
Moderate/heavy	124	38	33.3	1.25 (0.82 to 1.89)	1.28 (0.83 to 1.96)	
CVD mortality (n=208)						
Teetotallers	43	13	29.6	0.93 (0.51 to 1.69)	0.98 (0.53 to 1.82)	
Ex-drinkers	59	24	46.5	1.48 (0.92 to 2.37)	1.39 (0.86 to 2.26)	
Occasionals	199	62	31.8	1.00	1.00	
Light	230	62	26.7	0.84 (0.59 to 1.20)	0.94 (0.65 to 1.35)	
Moderate/heavy	124	47	41.4	1.31 (0.89 to 1.91)	1.34 (0.91 to 1.98)	
Non-CVD mortality (n=	86)					
Teetotallers	43	5	11.2	0.90 (0.34 to 2.37)	0.94 (0.34 to 2.55)	
Ex-drinkers	59	12	21.9	1.96 (0.97 to 3.94)	1.74 (0.84 to 3.63)	
Occasionals	199	23	11.9	1.00	1.00	
Light	230	32	13.8	1.14 (0.68 to 1.95)	1.38 (0.79 to 2.42)	
Moderate/ heavy	124	14	12.1	1.07 (0.55 to 2.07)	1.22 (0.62 to 2.41)	
All cause (n=294)						
Teetotallers	43	18	40.8	0.92 (0.55 to 1.53)	0.96 (0.57 to 1.62)	
Ex-drinkers	59	36	68.4	1.61 (1.09 to 2.38)	1.50 (1.00 to 2.24)	
Occasionals	199	85	43.7	1.00	1.00	
Light	230	94	40.5	0.93 (0.69 to 1.24)	1.05 (0.78 to 1.42)	
Moderate/ heavy	124	61	53.5	1.24 (0.89 to 1.73)	1.30 (0.93 to 1.83)	

*Age adjusted rates/1000 person years.

†Adjusted relative risks, by alcohol intake at Q5: A, age adjusted; B, adjusted for age, smoking, social class, BMI, pre-existing diabetes, stroke, and regular medication.

Table 2 Mortality from all causes, cardiovascular and non-cardiovascular disease in men with previous myocardial infarction (n=450) and men with angina only (n=200) at Q5, by alcohol intake at Q5

		All cause mortality			CVD mor	CVD mortality			Non-CVD mortality		
	Number of men	Number of cases	Rates*	Adjusted RR*	Number of cases	Rates*	Adjusted RR*	Number of cases	Rates*	Adjusted RR*	
Men with previou	s MI (n=4.	55)									
Teetotallers	33	14	46.4	1.00 (0.55 to 1.82)	11	36.4	1.07 (0.54 to 2.11)	3	10.0	0.82 (0.22 to 3.01)	
Ex-drinkers	47	31	80.5	1.75 (1.12 to 2.73)	22	57.1	1.63 (0.97 to 2.75)	9	23.4	2.18 (0.91 to 5.25	
Occasionals	139	62	45.3	1.00	48	35.0	1.00	14	10.3	1.00	
Light	153	70	47.2	1.09 (0.77 to 1.56)	47	31.7	0.95 (0.62 to 1.43)	23	15.5	1.74 (0.87 to 3.48)	
Moderate/heavy	83	45	61.2	1.50 (1.01 to 2.23)	37	50.3	1.50 (0.96 to 2.35)	8	10.9	1.45 (0.60 to 3.66)	
Men with angina	only (n=20	0)									
Teetotallers	10	4	35.1	0.97 (0.31 to 3.01)	2	17.5	0.70 (0.14 to 3.42)	2	17.6	1.61 (0.31 to 8.47)	
Ex-drinkers	12	5	42.8	0.94 (0.31 to 2.80)	2	17.1	0.79 (0.16 to 3.93)	3	25.7	1.09 (0.20 to 5.90	
Occasionals	60	23	39.4	1.00	14	24.0	1.00	9	15.4	1.00	
Light	77	24	30.3	1.03 (0.56 to 1.92)	15	18.9	1.06 (0.47 to 2.36)	9	11.4	0.97 (0.36 to 2.65	
Moderate/heavy	41	16	38.5	0.96 (0.48 to 1.91)	10	24.1	1.06 (0.43 to 2.63)	6	14.4	0.97 (0.32 to 2.95	

*Age adjusted rates/1000 person years.

†Relative risk (95% CI) adjusted for age, smoking, BMI, social class, pre-existing diabetes, stroke, and regular medication.

compared to occasional drinkers but these differences were not significant.

MEN WITH PREVIOUS MYOCARDIAL INFARCTION Of the 655 men with established CHD the majority had a history of myocardial infarction (n = 455). We examined the relation between alcohol intake and all cause mortality and cardiovascular mortality separately in men with myocardial infarction and men with angina only (n = 200). In both groups, there was little difference in outcome between lifelong teetotallers, occasional drinkers, and light drinkers. In men with previous myocardial infarction, ex-drinkers showed a significant increase in risk of all cause mortality and moderate/heavy drinking was associated with a significant increase in cardiovascular mortality (marginal) and all cause mortality compared to occasional drinking (table 2). The number of men in the angina only group was small and no consistent relation was seen with all cause mortality or cardiovascular mortality, but there appeared to be no adverse effect associated with moderate/ heavy drinking in men with angina only. There were only 57 deaths from non-cardiovascular causes in the myocardial infarction group but regular drinking (light or moderate/heavy) showed higher risk of non-cardiovascular deaths than occasional or lifelong teetotallers. No such increase in risk was seen for the angina only group (n = 19 deaths).

Table 3 Mortality from CHD, cardiovascular disease (CVD), and all causes in 655 men with established CHD at Q5 by smoking status at Q5 $\,$

Number of men	Number of cases	Rates*	Adjusted RR†
91	19	19.0	1.00
250	62	23.7	1.18 (0.70 to 2.00)
122	35	30.4	1.55 (0.87 to 2.74)
192	59	35.9	1.89 (1.11 to 3.22)
91	21	22.0	1.00
250	73	27.8	1.27 (0.77 to 2.08)
122	39	33.8	1.55 (0.90 to 2.66)
192	75	46.2	2.19 (1.33 to 3.60)
91	31	32.8	1.00
250	93	35.2	1.08 (0.72 to 1.64)
122	63	54.5	1.67 (1.08 to 2.60)
192	107	65.5	2.04 (1.35 to 3.09)
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*Age adjusted rates/1000 person years.

+Adjusted relative risk (95% CI) for age, smoking, social class, BMI, pre-existing diabetes, stroke, and regular medication.

CONTRAST WITH SMOKING CESSATION

In contrast to alcohol intake, current cigarette smoking was associated with a significant twofold increase in risk of mortality from CHD, cardiovascular disease, and all cause mortality in all men with established CHD (table 3). Long term ex-smokers showed mortality risks only slightly higher than those of never smokers. Those who had quit smoking within the last five years (Q1 to Q5 period) showed significantly increased risk of all cause mortality compared to never smokers but their levels of risk for CHD, cardiovascular mortality, and all cause mortality were lower than those who continued to smoke. All cause mortality in the recent ex-smokers was reduced by about 20%, cardiovascular mortality by 30%, and CHD mortality by 20%.

MEN WITH NO DIAGNOSED CHD

In men with no diagnosed CHD (n = 6514)there were 1064 deaths from all causes (12.7/1000 person years), including 327 CHD deaths (4.2/1000 person years). Compared to occasional drinking, regular drinking (light or moderate/heavy) was associated with significantly lower risk of CHD mortality but not of all cause mortality compared to occasional drinking after adjustment for age, social class, BMI, smoking, and diabetes. The adjusted relative risks (95% confidence interval (CI)) for CHD mortality for the five alcohol categories—lifelong teetotallers (n = 300), exdrinkers (n = 300), occasional drinkers (n = 1932), light drinkers (n = 2432), and moderate/heavy drinkers (n = 1550) were 0.70 (0.40 to 1.23), 1.10 (0.71 to 1.70), 1.00, 0.66 (0.50 to 0.87), and 0.73 (0.53 to 0.99), respectively. The corresponding relative risks (95% CI) for all cause mortality was 0.95 (0.70 to 1.29), 1.14 (0.87 to 1.49), 1.00, 0.89 (0.77 to 1.05), and 1.02 (0.86 to 1.21). The lower risk of CHD mortality associated with regular drinking persisted even after further exclusion of men with other cardiovascular related conditions (hypertension, diabetes, "other heart trouble", and stroke).

Discussion

While there is some evidence from clinical studies to suggest that heavy drinking is harmful in subjects with coronary artery disease,¹⁷

the effect of light to moderate drinking in such subjects has not been clearly established. The clinical studies suggest that alcohol may aggravate angina and that even low to moderate doses may impair left ventricular performance in patients with recent or old myocardial infarction.17 29 30 In our study involving middle aged and older men with established CHD, light alcohol intake (1-15 units per week) was not associated with any significant effect, deleterious or beneficial, compared to occasional drinkers (< 1 unit per week) or lifelong teetotallers. There is an indication that higher levels (\geq 3 drinks per day) may be harmful in those men with a history of previous myocardial infarction, which supports the findings of the clinical studies.¹⁷ This adverse effect at higher levels was seen even if heavy drinkers (> 6 drinks per day on a daily basis; > 42 units per week) were excluded. Some of those classified as light drinkers at Q5 might have been heavier drinkers in the past. This might have increased the risk of mortality in those classified as light drinkers. However, the confirmation in this study of the benefits of light to moderate drinking on CHD mortality in men with no diagnosis of CHD supports the validity of the findings in men with the diagnosis of CHD.

The effects of alcohol on subsequent risk of CHD has seldom been assessed in men with established CHD. Several prospective studies into alcohol related outcomes have examined the effects of alcohol in men with and without pre-existing disease (variously defined) and in most of these a U shaped or inverse relation has been observed between alcohol intake and outcome.10-16 In the recent US cancer prevention II study involving 490 000 men and women, using non-drinkers as the baseline, regular drinking was associated with a greater reduction in CHD mortality in those with cardiovascular related conditions, which included CHD, stroke, diabetes, and hypertension.¹⁵ These high risk subjects constituted one third of all subjects and contributed three quarters of all cardiovascular deaths. The reduction in risk was seen in those drinking less than daily but at least three times a week and was similar at all levels of alcohol intake-that is, there was no dose-response effect. It was suggested that alcohol may confer greater benefit on those at high risk of cardiovascular disease. This study did not examine specifically the outcome in men with established CHD (definite myocardial infarction or angina), who are at much higher risk of death than men with hypertension or diabetes. In our study, when we examined the effect of alcohol on CHD mortality in men with and without cardiovascular related conditions using similar criteria, the same pattern emerged in that the reduction in CHD mortality was greater in those with cardiovascular related conditions than in those without. However, when our analysis is restricted to men with established CHD no benefit is evident.

In contrast to our study, the recent US physicians health study showed that "men with a history of myocardial infarction with light to moderate intake of alcohol have a slight but clinically important decrease in total mortality compared with those who never or rarely drink alcohol".18 In that baseline group of never/ rarely drinkers at the time of inquiry, no distinction was made between lifelong teetotallers and ex-drinkers. In men aged 40-84 years usually drinking 2-6 drinks per week, total mortality was decreased by 28%, cardiovascular mortality by 24%, and noncardiovascular mortality by 39% after adjustment for age, smoking, diabetes, physical activity, and BMI. The subjects were elderly (69% were over 65 years old) and no account was taken of ill health or medication which might have led to reduction in alcohol intake other than for recall of hypercholesterolaemia or hypertension. The average intake in these high social class subjects was low and there were very few heavy drinkers in whom to examine the effects of heavier drinking (≥ 3 drinks per day) on mortality.

Critical to the comparison of the present study with previous studies is the issue of the baseline group used. In the physician health study, current non-drinkers constitute the baseline and we have shown that this mixed group of lifelong teetotallers and ex-drinkers is not appropriate for the evaluation of the effects of alcohol on cardiovascular or all cause mortality.8 23 24 There is little doubt that ex-drinkers have characteristics which greatly increase their mortality risk and they almost always have the highest mortality rates of any of the alcohol categories. Combining ex-drinkers and lifelong teetotallers, groups which vary in their proportions in different populations, can only lead to a base group with characteristics which cannot be adequately taken into account by adjustment procedures and which increase the risk of mortality.

In our present study, ex-drinkers had the highest risk of cardiovascular and all cause mortality compared to other groups, while lifelong teetotallers had a similar risk to occasional drinkers. Although light drinkers showed the lowest risk, the difference was very small compared to occasional drinkers. If the physician health study had used occasional drinkers (1-4 drinks per month) as their baseline comparison group, the reduction in total mortality in men with a previous myocardial infarction on 2-6 drinks per week would be 15%, for cardiovascular mortality the reduction would be 18%, and for non-cardiovascular mortality it would be 5%, none of which would be significantly different from baseline. If we had restricted the analyses to men with definite myocardial infarction and used all non-drinkers (lifelong teetotallers and ex-drinkers combined) as the comparison group, our results would be similar to those of the physicians health study, with both occasional drinkers (1-2 per month) and light drinkers showing lower mortality rates than non-drinkers. Our data suggest that the magnitude of benefit associated with light drinking (2-6 drinks per week) in the physicians health study has been exaggerated by the use of an inappropriate comparison group.

In our study, moderate/heavy drinking in men with a history of previous myocardial infarction is associated with a marginally significant increase in risk of cardiovascular death and a significant increase in all cause mortality. In the men with angina only and no history of myocardial infarction, albeit a very small group, there appears to be no adverse effect from moderate/heavy drinking. These findings may reflect the direct effect of alcohol on the damaged heart muscle of those with definite myocardial infarction, and may justify the comment that "in patients with pre-existing cardiovascular disease the use of alcohol is contraindicated, particularly where ventricular function is impaired".³¹ Our data suggest that light drinking may not be contraindicated but that moderate/heavy drinking may increase risk. In our moderate/heavy group, 84% were moderate drinkers with an intake of 16-42 UK units per week.

By contrast with the effects of alcohol, smoking cessation in men with established CHD was associated with a reduction in death from CHD, cardiovascular disease, and all causes, and for cardiovascular mortality the reduction was significant. Those who continued to smoke showed over a twofold increase in risk compared with those who had never smoked. Long term ex-smokers (who had given up for more than five years before the start of the follow up period) showed risk levels comparable to those of never smokers. Using the relative risks calculated in the cancer prevention II study, the authors have estimated the probability of death from any cause in the general US population aged 35-69 years.¹⁵ In both men and women, in drinkers and non-drinkers, smoking doubled the risk of death compared with non-smoking. The risk of death was slightly less in drinkers than non-drinkers; about 16% less in non-smokers and 6% less in smokers. They conclude that "the benefits of moderate alcohol consumption (1-2 drinks per day) are much smaller than the hazards of tobacco use".15

Conclusion

When an appropriate baseline group-that is, occasional drinkers-is used, regular light alcohol consumption (1-14 UK units per week) in men with established CHD is not associated with any significant benefit or deleterious effect for CHD, cardiovascular mortality, or all cause mortality. Higher levels of intake (≥ 3 drinks per day) are associated with increased mortality in men with previous myocardial infarction. By contrast, giving up smoking in men with established CHD halves the risk of mortality. Given the high absolute rate of death in men with established CHD, the absolute benefit of giving up smoking in men surviving a myocardial infarction is substantial. Time would be better spent assisting such men to stop smoking than trying to encourage the lifelong teetotallers or ex-drinkers among them to drink.

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- 1 Beaglehole R, Jackson R. Alcohol, cardiovascular diseases and all causes of death: a review of the epidemiological evi-dence. Drug Alcohol Rev 1992;11:275-90.
- . Demonstration of deductive meta 2 Maclure M ethanol intake and risk of myocardial infarction. Epidemiol
- *Rev* 1993;15:328–51. 3 Rimm EB, Klatsky A, Grobbee D, *et al.* Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine or spirits? BMJ 1996;312:731-6
- 4 Suh I, Shaten BJ, Cutler JA, et al. Alcohol use and mortality from coronary heart disease: the role of high-density lipoprotein cholesterol. Ann Intern Med 1992;116:881-7.
- 5 Goldberg RJ, Burchfiel, Reed DM, et al. A prospective study of the health effects of alcohol consumption in middle-aged and elderly men. Circulation 1994;89:651–9.
- 6 Rehm JT, Bondy SJ, Sempos CT, et al. Alcohol consumption and coronary heart disease morbidity and mortality. Am J Epidemiol 1997;**146**:495–501.
- Camargo CA, Hennekens C, Gaziano MJ, et al. Prospective study of moderate alcohol consumption and mortality in 7
- US male physicians. Arch Intern Med 1997;157:79–85. Wannamethee SG, Shaper AG. Lifelong teetotallers, exdrinkers and drinkers: mortality and incidence of coronary heart disease events in middle-aged British men. Int J Epidemiol 1997:26:440-2.
- Klatsky AL, Armstrong MA, Friedman GD. Relations of
- 9 Klatsky AL, Armstrong MA, Friedman GD. Relations of alcoholic beverage use to subsequent coronary artery disease hospitalisation. Am J Cardiol 1986;58:710-14.
 10 Klatsky AL, Armstrong MA, Friedman GD. Risk of cardio-vascular mortality in alcohol drinkers, ex-drinkers and non-drinkers. Am J Cardiol 1990;66:1237-42.
 11 Boffetta P, Garfinkel L. Alcohol drinking and mortality among men enrolled in an American Cancer Society
- among men enrolled in an American Cancer Society prospective study. *Epidemiology* 1990;1:342–8.
 12 Rimm EB, Giovannuci EL, Willett WC, *et al.* Prospective
- study of alcohol consumption and risk of coronary heart disease in men. Lancet 1991:86:381-2.
- Shaper AG, Wannamethee G, Walker M. Alcohol and
- coronardy heart disease: a perspective from the British regional heart study. *Int J Epidemiol* 1994;23:482–94.
 14 Doll R, Peto R, Hall E, *et al.* Mortality in relation to consumption of alcohol:13 years' observations on male British doctors. *BMJ* 1994;309:911–8.
- Thus M GOLOSE, *BM*, 1994;307:911–3.
 Thum MJ, Peto R, Lopez AD, *et al.* Alcohol consumption and mortality among middle-aged and elderly US adults. *N Engl J Med* 1998;337:1705–14.
 Shaper AG, Wannamethee G, Walker M. Alcohol and mor-
- tality in British men: explaining the U-shaped curve. Lancet 1988;ii:1268-73
- Ahlawat SK, Siwach SB. Alcohol and coronary artery disease. Int *J Cardiol* 1994;44:157–62.
 Muntwyler J, Hennekens CH, Buring JE, et al. Mortality and light to moderate alcohol consumption after myocar-
- dial infarction. Lancet 1998;**352**:1882–5. Shaper A G, Pocock S J, Walker M, et al. British regional
- heart study: cardiovascular risk factors in middle-aged men in 24 towns. BM7 1981;282:179-86.
- 20 Office of Population Censuses and Surveys. Social survey division. General household survey 1978. London: HMSO, 1980
- 21 Shaper AG, Pocock SJ, Ashby D, et al. Biochemical and haematological response to alcohol intake. Ann Clin Biochem 1985;22:50.
- 22
- Wannamethee G, Shaper AG. Changes in drinking habits in middle-aged British men. J R Coll Gen Pract 1988;38:440–2.
 Wanamethee G, Shaper AG. Men who do not drink: a report from the British regional heart study. Int J Epidemiol 23 1988:17:307-16.
- 24 Shaper AG, Wannamethee SG. The J-shaped curve and changes in drinking habit. In: Alcohol and cardiovascular dis-eases. Novartis Foundation symposium No 216. Chichester, UK: Wiley, 1998. 25 Walker M, Shaper AG. Follow-up of subjects in prospective
- studies in general practice. J R Coll Gen Pract 1984;34:365-
- 26 Walker M, Whincup PH, Shaper AG, et al. Validation of recall of doctor diagnosed heart attack and stroke: a postal
- questionaire and record review comparison. Am J Epidemiol 1998;148:355–61.
 Lampe FC, Walker MK, Lennon LT, et al. The validity of a self-reported history of doctor diagnosed angina. J Clin Epidemiol 1999;52:73–81.
- 28 Cox DR. Regression models and life tables (with discussion) 7 R Stat Soc B 1972;34:187-220.
- 29 Friedman HS. Acute effects of ethanol on myocardial blood flow in the non-ischaemic and ischaemic heart. Am J Car*diol* 1981;47:61–7. Siwach SB, Gupta SP, Lohan A. Acute effects of ethyl alco-
- 30 hol on left ventricular performance in myocardial infarction cases-a study of systolic time intervals. J Assoc Phys Ind 1985;33:389-91.
- 31 Preedy VR, Richardson PJ. Ethanol induced cardiovascular disease. Br Med Bull 1994;50:152-63