

Alcohol consumption, diet, coronary risk factors, and prevalent coronary heart disease in men and women in the Scottish heart health study

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Abstract

Study objective – To measure the relationship between reported alcohol consumption and prevalent diagnosed and undiagnosed coronary heart disease (CHD) in men and women to see how much could be explained by covariation with diet, lifestyle, and biomedical factors.

Design and setting – This was a cross sectional, random population survey covering 22 districts of Scotland and using general practitioner patient lists as the sampling frame. Odds ratios for prevalent CHD at different levels of alcohol consumption taken from a seven day recall were analysed. These ratios were then adjusted for lifestyle and biomedical factors.

Participants – Male and female responders aged 40–59 years who completed the survey questionnaire and attended the survey clinic.

Main results – The participation rate of those invited was 74%. Of the 10 359 responders, 658 were excluded because of missing alcohol data or ambiguous cardiovascular status. The questionnaire was used to designate 7058 drinkers and 2643 non-drinkers, who were then classified as having diagnosed or undiagnosed CHD, or who were controls. The prevalence of diagnosed CHD decreased with increasing alcohol consumption while undiagnosed CHD had a “U” shaped relationship. Patterns were similar in men and women if allowance was made for the lower alcohol consumption in women. Adjustment for several diet and lifestyle factors and for additional biomedical factors reduced the apparent protective effect of alcohol, leaving a modest but statistically insignificant ($p > 0.05$) reduction in CHD prevalence among light to moderate consumers compared with those who drank no alcohol. Wine drinkers seemed to be at lower risk than beer drinkers in both sexes.

Conclusions – These results tend to confirm that intermediate alcohol consumption is a component and contributor to a low coronary risk lifestyle. Its effects are largely explained by adjusting for both confounding lifestyle associations and for biomedical effects but the remaining effect, and the lower risk with wine drinking compared with beer, are intriguing.

Advice on alcohol habits should not be determined solely by the moderate apparent benefit to risk of CHD, however, as other disease risks cannot be ignored.

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Numerous reports from various countries – the USA (Framingham,¹ MRFIT,² NHANES,³) to Britain (British regional heart study,⁴ Whitehall,⁵) France,⁶ Yugoslavia,⁷ Australia,⁸ and Hawaii⁹ – have shown that moderate alcohol consumption, in comparison with abstinence, has an apparent protective effect on the incidence or mortality from coronary heart disease (CHD). Shaper, based on his findings from the British regional heart study,¹⁰ has claimed that the association is indirect and not causal and results from a change in alcohol consumption towards abstinence as disease becomes established. Several investigators, however, have now produced consistent evidence from studies in which lifelong non-drinkers have been distinguished from others, and some claim that the link is indeed causal.^{11–16} Interest is now focused on examining other confounding factors in lifestyle,¹⁷ the biomedical consequences of increased ethyl alcohol consumption, such as increasing HDL cholesterol,^{2,18–20} decreasing platelet aggregation,²¹ other haemostatic factors,²² or other components of drinks, such as antioxidant micronutrients in red wine.^{23–24}

Unlike many earlier studies, the Scottish heart health study (SHHS) included information on aspects of diet, through a food frequency questionnaire, while recording current alcohol consumption through seven day recall. It also has data on most of the established CHD risk factors that have been suggested as confounders or mediators in the alcohol-CHD relationship. The SHHS has the further advantages of being a large population drawn randomly from the general population which includes roughly equal numbers of men and women. Not only is there less information on women,^{25–27} but few other reports have been able to compare men and women directly within the same study.^{3,8,15,28} Unlike southern European cultures in which alcohol is generally consumed, the Scots have a Calvinist tradition in which the drinking of alcohol is discouraged on traditional moral grounds. Hence, despite

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their reputation for heavy drinking, many Scots drink little or no alcohol, so the range of consumption is broad. For these reasons the SHHS data have been explored to assess the relationship between alcohol consumption and prevalent CHD and to consider how far this is explained by covariation with other factors.

Materials

The SHHS recruited a random sample of 10 359 men and women between the ages of 40 and 59 years from 22 districts across Scotland during 1984–6. They completed a postal, self administered health record which included questions on basic demographic details, employment, current medication, past medical history, smoking, and physical activity. The OPCS definition of occupational social class²⁹ was used in this study; the unemployed were classified by their last job and married women by their husband's job. Physical activity questions asked whether participants were active, average, or inactive at leisure and at work. The health record incorporated the Rose chest pain questionnaire³⁰ and a food frequency questionnaire.³¹ The latter asked about the frequency of consumption of 50 food items or groups and the family's weekly consumption of butter, margarine, vegetable oils, cheese, cream, and sugar. For alcohol, each respondent was asked to recall how many pints of beer, glasses of wine, or glasses of spirits he or she had consumed on each of the previous seven days, and this was converted to units. Mean daily nutrient intakes were estimated using standard UK food tables;³² total energy was calculated by including alcohol. Intake of the three antioxidant vitamins was summarised by their composite principal component (PC) score.³³ Further details of the dietary assessments are given elsewhere.³⁴

Participants were invited to bring their health record with them to a survey clinic where it was checked. Their blood pressure, height, and weight were measured and a 12-lead electrocardiogram was recorded. A blood sample was taken from which total cholesterol, HDL cholesterol, triglycerides, fibrinogen and γ -glutamyltransferase (GGT) were estimated. Detailed methods and main results have been published elsewhere.^{35–37}

Methods

One disadvantage of cross sectional data used here is that alcohol consumption (and other aspects of lifestyle) may change as a result of a diagnosis of CHD. Indeed, this is the basis of Shaper's explanation¹⁰ of previously observed alcohol-CHD relationships. To address this problem and to explore Shaper's hypothesis, prevalent CHD was defined in two exclusive groups. *Diagnosed CHD* was defined as a medical history of diagnosed angina or myocardial infarction, as reported in the SHHS questionnaire. Subjects who gave positive answers to the Rose chest pain questionnaire or showed Q/QS or ST or T wave findings on the electrocardiogram³⁸ without a history of medical

diagnosis were considered to have *undiagnosed CHD*. The two prevalence groups were compared separately with the *control* group – those with no history or indications of CHD. Anyone who would otherwise have been classed as a control but who nonetheless reported current consumption of drugs listed as cardiovascular in the *British National Formulary*, however,³⁹ was excluded from the analysis as they were considered to have ambiguous status in respect of CHD.

Total alcohol consumption was related to CHD prevalence by defining five, mutually exclusive consumption groups. Non-drinkers (over the seven days) were kept in a separate category. The remainder (for each sex) were split into four groups, as near constant size as possible to maximise statistical efficiency, using their weekly consumption in alcohol units (table 1). The groupings used by others, such as for the general household survey, were too unequal to be useful. For a subsidiary analysis, pure spirits and wine drinkers were compared with beer drinkers: those who mixed their drinks were omitted.

Alcohol consumption was related to other CHD risk factors by comparing mean values or proportions across the alcohol consumption groups. The ungrouped alcohol data were compared with each quantitative risk factor through Spearman's rank correlation coefficients (tables 2 and 3). Rank correlations were used instead of the more usual Pearson correlations, because the distribution of alcohol consumption is extremely skewed.

The alcohol-CHD relationship, adjusted for five sets of risk factors, was then estimated using linear logistic regression (tables 4 and 5 and figure). All adjustment sets include age. The first set contained no other variables. The second set added HDL cholesterol, the variable most often cited as the link between alcohol and CHD. The third adjustment set added body mass index, diastolic blood pressure, total cholesterol, triglycerides, and fibrinogen (risk set "A"). The fourth (risk set "B") comprised age plus potential confounding lifestyle factors, occupational social class, activity in leisure and at work, cigarette smoking status, and antioxidant vitamin, fat, saturated fat, fibre, and total energy consumption. Since a large dietary intake may include large amounts of all food-stuffs, including alcohol, total energy consumption was included as an adjustment for the total size of the diet. The final adjustment set had all variables. This certainly gives rise to over adjustment, since many variables may themselves be modified by alcohol consumption.

In all logistic regression analyses each quantitative risk factor besides age and cigarette consumption was divided into six groups: five equal fifths (about the quantiles) plus those with a missing value for that variable. Age was left as a continuous variable (no missing values) and cigarette smoking status was defined using six groups: three graded groups for current consumption (1–14, 15–24, and over 24 cigarettes per day), never smokers, ex-smokers, and missing values.

Table 1 Coronary heart disease (CHD) status in relation to alcohol consumption group

Alcohol group (U/wk)	CHD group			
	Diagnosed	Undiagnosed	Control	Total
Men:				
0	96 (12%) [10%]	142 (17%) [15%]	686 [74%]	924 [100%]
1-7	83 (10%) [8%]	130 (14%) [13%]	779 [79%]	992 [100%]
8-15	76 (9%) [8%]	126 (14%) [13%]	768 [79%]	970 [100%]
16-29	66 (8%) [7%]	131 (14%) [13%]	798 [80%]	995 [100%]
30 and over	61 (7%) [6%]	153 (16%) [15%]	787 [79%]	1001 [100%]
Total	382 (9%) [8%]	682 (12%) [14%]	3818 [78%]	4882 [100%]
Women:				
0	127 (9%) [7%]	314 (20%) [18%]	1278 [74%]	1719 [100%]
1-2	27 (5%) [4%]	137 (20%) [19%]	553 [77%]	717 [100%]
3-5	32 (4%) [4%]	139 (16%) [16%]	720 [81%]	891 [100%]
6-9	30 (5%) [4%]	99 (14%) [13%]	607 [82%]	736 [100%]
10 and over	23 (4%) [3%]	116 (16%) [15%]	617 [82%]	756 [100%]
Total	239 (6%) [5%]	805 (18%) [17%]	3775 [78%]	4819 [100%]

Note – percentages in round brackets are relative to the stated group plus the control group only; percentages in square brackets are overall percentages.

Results

Of the 10 359 responders (74% of those invited), 619 were excluded as they were considered to be of ambiguous CHD status – they were on cardiovascular medication without a history of CHD. The pattern of alcohol consumption in these 619 was similar to that in those with undiagnosed CHD: 23% of men and 39% of women were non-drinkers, compared with 21% and 39% respectively in the

undiagnosed group. A further 39 subjects were excluded from analysis because they failed to answer the seven day alcohol recall questions (two of the 619 excluded because of CHD medication also had missing data on alcohol). The remaining 9701 subjects are classified in table 1 in relation to their alcohol consumption (in units over seven days) and their CHD status.

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Table 2 Rank correlations with alcohol, means (SD) or percentages (SE) and tests of significance to compare alcohol groups for men

Variable	Rank correlation	Alcohol group (U/wk)					p value
		0	1-7	8-15	16-29	30+	
HDL cholesterol (mmol/l)	0.34	1.22 (0.31)	1.26 (0.32)	1.32 (0.31)	1.44 (0.37)	1.55 (0.41)	‡
Total energy (kcal/d)*	0.26	2253 (624)	2204 (529)	2242 (555)	2360 (575)	2726 (657)	‡
Cigarettes (no/d)†	0.15	7.2 (11.4)	5.7 (10.1)	7.4 (11.3)	7.9 (11.5)	11.8 (13.7)	‡
Diastolic blood pressure (mm Hg)	0.10	83.7 (11.8)	82.0 (10.9)	83.5 (11.7)	84.8 (11.4)	86.1 (11.8)	‡
Triglycerides (mmol/l)	0.003	2.35 (1.40)	2.25 (1.24)	2.26 (1.32)	2.32 (1.45)	2.41 (1.53)	0.10
Fat (g/d)*	0.003	93.9 (30.9)	89.1 (25.2)	89.1 (27.3)	90.6 (27.1)	93.8 (29.7)	‡
Total cholesterol (mmol/l)	0.001	6.38 (1.20)	6.35 (1.19)	6.36 (1.16)	6.40 (1.14)	6.37 (1.19)	0.94
Saturated fat (g/d)*	-0.008	41.6 (15.1)	39.4 (12.7)	39.6 (13.6)	39.8 (13.5)	41.3 (14.5)	0.004
Antioxidant vitamin PC*	-0.03	1280 (910)	1328 (836)	1339 (850)	1239 (816)	1236 (853)	0.02
Body mass index (kg/m ²)	-0.03	26.3 (3.7)	26.0 (3.2)	25.9 (3.4)	25.9 (3.4)	25.9 (3.5)	0.02
Fibrinogen (g/l)	-0.04	2.39 (0.71)	2.27 (0.64)	2.29 (0.64)	2.28 (0.72)	2.30 (0.71)	0.003
Fibre (g/d)*	-0.12	21.7 (8.2)	22.5 (7.5)	21.9 (7.2)	20.7 (7.2)	19.6 (7.0)	‡
Age (y)	-0.13	50.5 (5.8)	50.3 (5.7)	49.5 (5.7)	49.0 (5.7)	48.4 (5.6)	‡
Active in work (%)		45 (2)	39 (2)	40 (2)	42 (2)	46 (2)	0.003
Active in leisure (%)		22 (1)	26 (1)	23 (1)	24 (1)	20 (1)	0.06
Non-manual occupation (%)		37 (2)	54 (2)	49 (2)	44 (2)	32 (1)	‡
Ex-smokers (%)		18 (1)	22 (1)	20 (1)	21 (1)	19 (1)	0.06

* Estimated from the food frequency questionnaire; † Kruskal-Wallis test used.

‡ = p < 0.001.

PC = principal component score.

Table 3 Rank correlations with alcohol, means (SD) or percentages (SE) and tests of significance to compare alcohol groups for women

Variable	Rank correlation	Alcohol group (U/wk)					p value
		0	1-2	3-5	6-9	10+	
HDL-cholesterol (mmol/l)	0.21	1.59 (0.40)	1.65 (0.39)	1.68 (0.38)	1.73 (0.41)	1.87 (0.48)	‡
Total energy (kcal/d)*	-0.004	1771 (488)	1775 (454)	1740 (465)	1759 (460)	1795 (497)	0.20
Cigarettes (no/d)†	0.07	6.4 (9.5)	4.2 (7.8)	5.5 (8.8)	6.5 (9.3)	9.0 (11.1)	‡
Diastolic blood pressure (mm Hg)	-0.03	81.4 (11.3)	80.2 (10.9)	79.4 (10.9)	80.4 (11.9)	81.5 (12.3)	‡
Triglycerides (mmol/l)	-0.10	1.74 (1.03)	1.61 (0.96)	1.50 (0.83)	1.59 (0.95)	1.59 (1.10)	‡
Fat (g/d)*	-0.08	79.7 (26.1)	79.4 (23.4)	76.7 (23.6)	76.5 (24.7)	74.4 (24.9)	‡
Total cholesterol (mmol/l)	-0.07	6.7 (1.3)	6.6 (1.3)	6.4 (1.3)	6.5 (1.3)	6.4 (1.3)	‡
Saturated fat (g/d)*	-0.09	36.3 (13.0)	36.1 (11.9)	34.9 (12.0)	34.5 (12.3)	33.4 (12.3)	‡
Antioxidant vitamin PC*	0.008	1264 (892)	1381 (826)	1268 (838)	1274 (807)	1254 (825)	0.02
Body mass index (kg/m ²)	-0.08	26.1 (4.9)	25.4 (4.5)	25.0 (3.9)	25.1 (4.3)	25.0 (4.0)	‡
Fibrinogen (g/l)	-0.11	2.45 (0.73)	2.36 (0.67)	2.35 (0.64)	2.34 (0.68)	2.25 (0.62)	‡
Fibre (g/d)*	-0.01	19.0 (7.4)	20.6 (7.4)	19.4 (6.7)	19.1 (6.7)	18.6 (7.1)	‡
Age (y)	-0.15	50.3 (5.8)	49.9 (5.7)	48.7 (5.6)	48.6 (5.8)	48.0 (5.4)	‡
Active in work (%)		47 (1)	49 (2)	48 (2)	49 (2)	47 (2)	0.85
Active in leisure (%)		19 (1)	21 (2)	19 (1)	19 (1)	19 (1)	0.85
Non-manual occupation (%)		36 (1)	52 (2)	52 (2)	52 (2)	54 (2)	‡
Ex-smokers (%)		16 (1)	19 (1)	25 (1)	24 (2)	26 (2)	‡

* Estimated from the food frequency questionnaire; † Kruskal-Wallis test used.

‡ = p < 0.001.

PC = principal component score.

sexes differed, the class intervals were different: men had much higher class midpoints. While in men the number of non-drinkers was similar to the number in each of the four "quarter" groups of drinkers, non-drinkers accounted for over 35% of all women. Table 1 shows that the prevalence of diagnosed CHD decreased with increasing alcohol consumption. The prevalence of undiagnosed CHD was lowest in the middle alcohol groups, but increased at both ends of the consumption spectrum, following a "U" shaped curve.

χ^2 tests were used to compare the distributions of alcohol consumption in the diagnosed and undiagnosed CHD groups only. The differences were highly significant ($p=0.002$) for women, but more marginal for men ($p=0.07$). This was considered sufficient evidence to justify analysing separately diagnosed CHD versus controls and undiagnosed CHD versus controls.

In table 2 (men) and table 3 (women) alcohol consumption is related to 17 other risk factors for CHD. Mean (SD) values are given for all quantitative risk factors, together with an F test of equality of means. For current cigarette consumption, the equivalent Kruskal-Wallis non-parametric test was used because this variable is extremely skewed. Cigarette con-

sumption means are still shown because the corresponding medians are virtually always zero. For the percentage variables (occupying the bottom four lines of each table) SE are given, together with χ^2 tests of equality of percentages. The tables give percentages of ex-smokers; for non-drinkers 69% of men and 54% of women were ever-smokers of cigarettes, and for drinkers the corresponding percentages were 75% and 61% respectively.

Significant differences ($p<0.05$) between the means were found in almost every case. These sometimes corresponded to small absolute differences, however, and reflected the large sample size rather than any biological significance. For several variables the order of magnitude of the means followed a monotonic increase or decrease, or the shape of an upright or upside down "U", across the alcohol groups. For men the most favourable values of the risk factor almost always occurred in the 1-7 units/week alcohol group. The non-drinkers or the group who consumed ≥ 30 units almost always had the least favourite values. For women the picture is more complex: the 1-2, 3-5, and ≥ 10 units/week groups all had a substantial number of "best" outcomes. The "worst" was always either the non-drinkers or the highest consumption group. For each sex, HDL cho-

Table 4 Odds ratios in relation to alcohol group (base = non-drinkers), 95% confidence intervals and tests for polynomial effects of alcohol for men

Alcohol group (U/wk)	Adjusted for				
	Age	Age, HDL cholesterol	Risk set A*	Risk set B*	Risk sets A+B
Diagnosed coronary heart disease:					
1-7	0.78 (0.57, 1.07)	0.83 (0.63, 1.15)	0.88 (0.64, 1.22)	0.88 (0.63, 1.23)	0.99 (0.70, 1.41)
8-15	0.79 (0.57, 1.09)	0.92 (0.66, 1.27)	0.93 (0.66, 1.30)	0.83 (0.59, 1.18)	0.99 (0.69, 1.42)
16-29	0.71 (0.51, 0.99)	0.91 (0.64, 1.28)	0.85 (0.59, 1.21)	0.79 (0.55, 1.14)	0.97 (0.66, 1.43)
30 and over	0.71 (0.50, 0.99)	0.98 (0.68, 1.40)	0.93 (0.64, 1.36)	0.77 (0.51, 1.15)	1.04 (0.67, 1.61)
Linear	0.08	0.75	0.22	0.75	0.99
Quadratic	0.24	0.58	0.44	0.44	0.75
Higher order	0.54	0.61	0.91	0.82	0.99
Undiagnosed coronary heart disease:					
1-7	0.81 (0.62, 1.04)	0.82 (0.63, 1.06)	0.86 (0.66, 1.12)	0.88 (0.68, 1.15)	0.91 (0.69, 1.19)
8-15	0.82 (0.63, 1.07)	0.86 (0.66, 1.12)	0.85 (0.65, 1.13)	0.87 (0.66, 1.14)	0.88 (0.67, 1.16)
16-29	0.84 (0.65, 1.09)	0.93 (0.71, 1.22)	0.87 (0.66, 1.14)	0.84 (0.64, 1.10)	0.85 (0.64, 1.19)
30 and over	1.03 (0.80, 1.32)	1.19 (0.92, 1.55)	1.05 (0.80, 1.38)	0.86 (0.64, 1.14)	0.86 (0.63, 1.17)
Linear	0.28	0.02	0.40	0.32	0.40
Quadratic	0.07	0.11	0.34	0.10	0.40
Higher order	0.45	0.41	0.82	0.71	0.95

* Risk set A: age, total cholesterol, HDL cholesterol, diastolic blood pressure, triglycerides, fibrinogen, body mass index; risk set B: age, social class, activity in work, activity in leisure, cigarette smoking status, antioxidant PC, fat consumption, saturated fat consumption, fibre consumption, total energy consumption.

Table 5 Odds ratios in relation to alcohol group (base = non-drinkers), 95% confidence intervals and tests for polynomial effects of alcohol for women

Alcohol group (U/wk)	Adjusted for				
	Age	Age, HDL cholesterol	Risk set A*	Risk set B*	Risk sets A+B
Diagnosed coronary heart disease:					
1-2	0.51 (0.33, 0.78)	0.52 (0.34, 0.80)	0.56 (0.36, 0.86)	0.54 (0.35, 0.85)	0.57 (0.36, 0.91)
3-5	0.52 (0.35, 0.78)	0.56 (0.37, 0.84)	0.62 (0.41, 0.95)	0.55 (0.36, 0.83)	0.62 (0.41, 0.96)
6-9	0.60 (0.40, 0.91)	0.68 (0.45, 1.04)	0.71 (0.46, 1.08)	0.66 (0.43, 1.01)	0.74 (0.47, 1.15)
10 and over	0.49 (0.31, 0.77)	0.61 (0.38, 0.97)	0.60 (0.37, 0.97)	0.50 (0.31, 0.82)	0.61 (0.37, 1.01)
Linear	0.0007	0.02	0.003	0.03	0.05
Quadratic	0.02	0.03	0.07	0.09	0.15
Higher order	0.06	0.06	0.09	0.12	0.14
Undiagnosed coronary heart disease:					
1-2	1.02 (0.82, 1.28)	1.04 (0.83, 1.31)	1.11 (0.88, 1.39)	1.12 (0.88, 1.41)	1.17 (0.92, 1.48)
3-5	0.84 (0.67, 1.04)	0.87 (0.69, 1.08)	0.94 (0.75, 1.17)	0.94 (0.74, 1.18)	1.02 (0.80, 1.28)
6-9	0.71 (0.55, 0.91)	0.75 (0.58, 0.96)	0.77 (0.60, 1.00)	0.79 (0.61, 1.02)	0.84 (0.65, 1.09)
10 and over	0.84 (0.66, 1.06)	0.92 (0.72, 1.17)	0.91 (0.71, 1.17)	0.91 (0.71, 1.18)	0.96 (0.74, 1.24)
Linear	0.03	0.18	0.19	0.17	0.37
Quadratic	0.05	0.07	0.32	0.25	0.65
Higher order	0.33	0.32	0.20	0.18	0.14

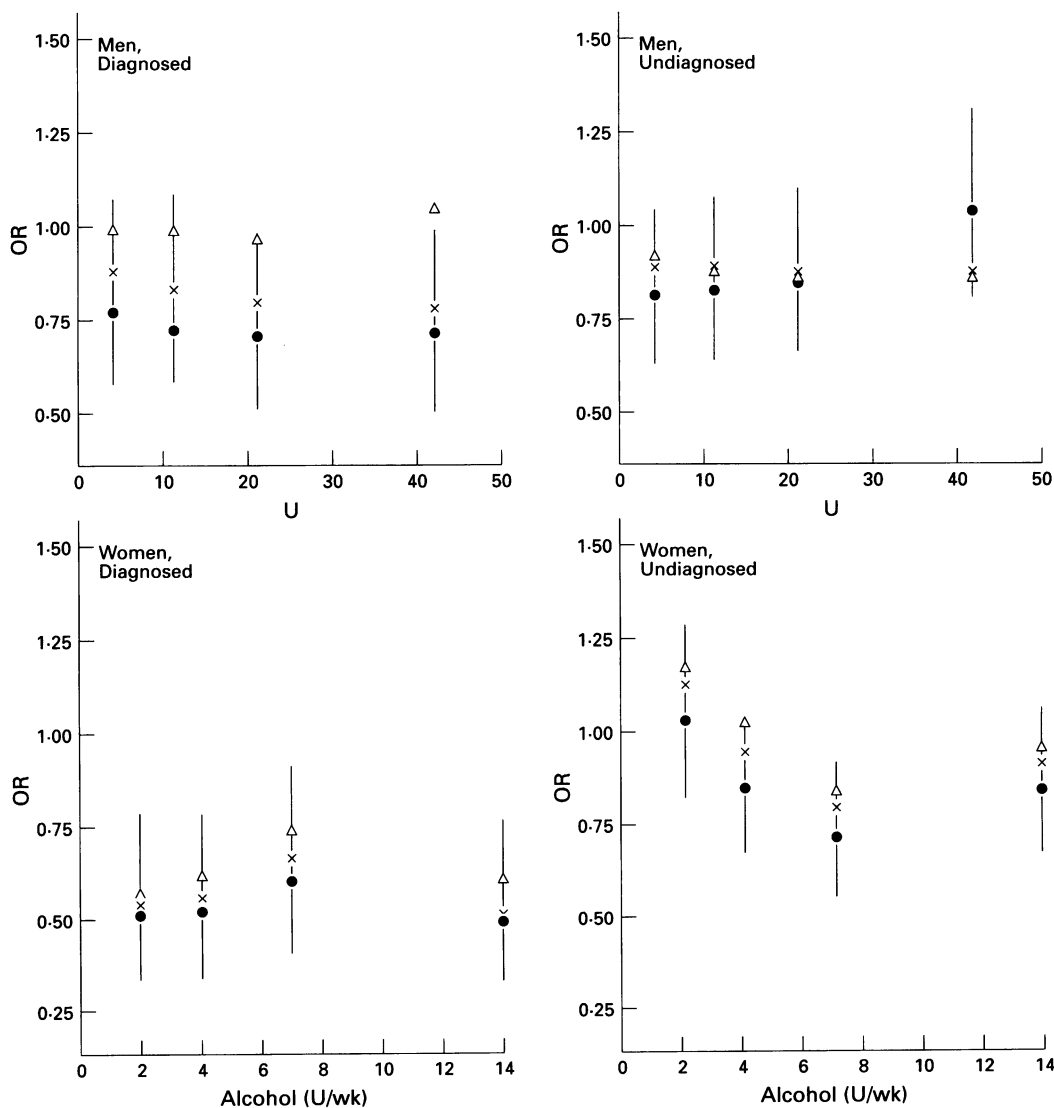
* Risk set A: age, total cholesterol, HDL cholesterol, diastolic blood pressure, triglycerides, fibrinogen, body mass index; risk set B: age, social class, activity in work, activity in leisure, cigarette smoking status, antioxidant PC, fat consumption, saturated fat consumption, fibre consumption, total energy consumption.

lesterol was most highly positively correlated with alcohol, and age was most highly negatively correlated. Otherwise there were substantial differences between the sexes: many correlations change sign (even if close to zero) when switching sex. The greatest change was for total energy consumption which was highly positively correlated with alcohol consumption for men but showed a very slight negative correlation for women. Note that the variables were ordered by decreasing magnitude of the correlation for men in both tables 2 and 3.

Tables 4 and 5 show adjusted odds ratios (OR) with 95% confidence intervals (CI) for prevalent diagnosed and undiagnosed CHD separately, for each risk set. The non-drinkers were taken as the base group (OR=1) in each comparison. The figure illustrates three of the risk sets. The median consumption in each alcohol group is plotted on the horizontal axes and 95% CI are included only for the initial adjustment (for age alone). The full adjustment

in the tables and figures adjusts for all the variables listed in tables 2 and 3.

For men, after adjustment only for age, the prevalence of diagnosed CHD decreased with increasing alcohol consumption, but the prevalence of undiagnosed CHD decreased and then increased in the highest consumption group. The non-drinking group had a substantially higher OR of diagnosed CHD than all the drinking groups. The drinking groups themselves had similar ORs for diagnosed CHD. For undiagnosed CHD, the zero and highest alcohol consumption groups had similar ORs; the remaining groups had lower but virtually constant ORs. Adjustment for potential confounders accentuated the linear decrease for diagnosed CHD and removed the upturn in the highest consumption group for undiagnosed CHD. Full adjustment removed any effect of alcohol upon diagnosed CHD but left a pattern of slightly decreasing ORs for undiagnosed CHD. The proportion of the



Male and female odds ratios (OR) for diagnosed and undiagnosed coronary heart disease (CHD) plotted against the median of each alcohol group. For definitions of alcohol groups see table 1. Vertical lines give the 95% confidence intervals for the age adjusted odds ratios. ● Adjusted for age; × Adjusted for age, occupational social class, activity in work, activity in leisure, cigarette smoking status, antioxidant principal component score, fat, saturated fat, fibre, and total energy consumption; △ Adjusted as above, plus total cholesterol, HDL cholesterol, diastolic blood pressure, triglycerides, fibrinogen, and body mass index.

effects of alcohol on CHD risk accounted for by lifestyle alone was 59% and metabolic factors alone 82% in diagnosed CHD, while the figures were 69% and 30% respectively in undiagnosed CHD. There was no significant interaction ($p > 0.05$) of alcohol and age upon diagnosed or undiagnosed CHD, before or after full adjustment.

For women the patterns after adjustment for age alone were very similar to those for men, except that the difference between non-drinkers and drinkers for diagnosed CHD was more pronounced and the "U" shaped pattern for undiagnosed CHD was sharper and more skewed. As was the case for men, adjustment progressively increased the ORs in all drinking groups. Extra adjustment for women left the initial patterns essentially the same, except that the OR for the 1–2 units/week category rose above unity. The contribution of lifestyle factors alone to diagnosed CHD was 26% and metabolic factors 46%, whereas for undiagnosed CHD the figures were 45% and 38% respectively. As with men, there were no significant interactions of alcohol and age upon CHD.

Tests of significance were applied to look for linear trends, quadratic or higher order (cubic or above) polynomial effects, and these are reported in tables 4 and 5. For men, after adjustment only for age, there was some evidence of a negative linear trend ($p = 0.08$) in diagnosed CHD with alcohol consumption, and evidence of a quadratic pattern ($p = 0.07$) for undiagnosed CHD. Further adjustment rendered all polynomial effects non-significant ($p \geq 0.10$), except for evidence of linearity after extra adjustment for HDL cholesterol in diagnosed CHD. For women, after adjustment only for age, the ORs for diagnosed CHD had a significant linear ($p = 0.0007$) and quadratic ($p = 0.02$) pattern but higher order polynomial effects were also important ($p = 0.06$). ORs for undiagnosed CHD had a significant linear ($p = 0.03$) and quadratic ($p = 0.05$) component only. Adjustment for confounding factors reduced the significance of the linear and quadratic effects for diagnosed CHD. Full adjustment left only the linear trend significant ($p = 0.05$). All adjustments in addition to age removed all significance for undiagnosed CHD, although adjustment for age and HDL cholesterol only left the quadratic effect just marginally non-significant ($p = 0.07$).

Implicit in the calculation of alcohol consumption in units used in the SHHS and in other studies is the assumption that it is the ethyl alcohol component which is at the centre of the risk and benefit associations. Recent interest in micronutrients^{23–24} present in red wine has complicated this assumption. A subsidiary analysis of ORs comparing spirits and wine drinkers (red was not distinguished) with beer drinkers showed some interesting tendencies, although the small numbers involved meant that most of the ORs were not significantly different from unity, and we are therefore not reporting these in detail.

Compared with beer drinkers, the age adjusted OR for diagnosed CHD in men was increased in spirits drinkers to 1.24 and un-

changed by the further multiple adjustments in these analyses, whereas in wine drinkers the OR was 0.72, multiple full adjustment bringing it up to 0.87. For undiagnosed CHD in men, spirits and wine drinkers had marginally reduced ORs compared with beer drinkers and further adjustment raised them. In women both spirits and wine consumption, compared with beer, were associated with considerably reduced age adjusted ORs (0.52 for spirits, 0.34 for wine) for diagnosed CHD but less so (0.78 and 0.69) for undiagnosed CHD. Multiple full adjustment raised these ORs only partly towards unity. The tendencies for the different alcoholic drinks in the four categories of end points (diagnosed and undiagnosed CHD, men and women) were not entirely consistent but wine seemed to be associated with a lower risk than beer in all four categories, and particularly so in women and those with diagnosed CHD.

Discussion

In this analysis current alcohol consumption has been shown to be related to several other variables which are frequently proposed as causal factors for CHD (and other diseases). Judged in terms of optimum values, non-drinkers and heavy drinkers generally tend to have the least healthy lifestyle, while light and/or moderate drinkers have the most healthy overall lifestyle profile (tables 2 and 3). Of the many risk factors considered here, HDL cholesterol had the strongest correlation with alcohol consumption – mean values increased consistently across the consumption groups. Previous work on the SHHS⁴⁰ has shown that alcohol is the most important dietary predictor of HDL cholesterol. It is claimed by some workers that HDL cholesterol mediates a large proportion of alcohol associated coronary prevention,²⁰ although others have been more cautious.⁴¹ Recently confirmed low levels of coronary heart disease in France⁴² were not associated with higher levels of HDL cholesterol than are seen in other countries,²¹ despite the high alcohol consumption of the French, so a single factor approach based on HDL cholesterol has limitations.

In this report alcohol consumption and most of the covariates have been considered in grouped form rather than in their original discrete or continuous forms. This has been done for three reasons. Firstly, there is no reason to suppose a linear or low order polynomial relationship between alcohol and CHD, indeed the findings here agree with previous studies which suggest a non-symmetric, non-linear relationship, and the relationship may even vary in different age and sex groups. Similarly, the use of categories allows for greater flexibility in modelling covariation. Secondly, the grouped format allows missing values to be included as a separate category: although the inclusion of these missing data does give a different interpretation to the underlying model. Thirdly, grouped results in such a large study are rather easier to interpret, for example through displaying risk factor values in relation to alcohol consumption groups. Another question is

whether our use of five groups in most cases was appropriate. Fewer groups would have provided less information about both the alcohol-CHD pattern and the effect of co-variation; more would have had the reverse effect but would also have meant both wider CIs for the ORs and larger, less easy to read tables. Five groups seemed to be the best compromise in this case.

When relating alcohol consumption to CHD prevalence in this study, diagnosed and undiagnosed CHD were considered separately in case knowledge of the diagnosis had precipitated a lowering of alcohol consumption as Shaper has suggested.¹⁰ We have used this approach in previous cross sectional analyses of the SHHS data.³³ In the main, undiagnosed CHD had a "U" shaped relationship with alcohol, whereas diagnosed CHD had a decreasing linear trend. The heterogeneity between these is consistent with, but not proof of, a change in alcohol habit after CHD diagnosis, as the undiagnosed group included milder cases and could have been distorted by numbers of false positives. This is because some of the ECG criteria and the Rose questionnaire are not highly specific for CHD.

Overall, the effect of alcohol on CHD prevalence was small, especially in the undiagnosed CHD group. Adjustment for other cardiovascular risk factors reduced the difference between non-drinkers and each drinking group in a progressive fashion. Despite the lack of a strong effect of alcohol on CHD, there was only one case in which the prevalence was substantially higher in any drinking group compared with non-drinkers. This was for very occasional (1-2 units/week) female drinkers, and even then was seen only after considerable adjustment (the fully adjusted OR was 1.17 with a 95% CI of 0.92, 1.48). With this exception, full adjustment (what we consider to be over adjustment) still left alcohol showing a protective effect or (among diagnosed men) no effect. Taking the adjustment for lifestyle factors (risk set B) on undiagnosed CHD as the most reliable evidence, the third highest groups (16-29 units/week in men; 6-9 units/week in women) had the best outcomes.

Drinking habits were measured over a recent seven day period in this study. This is not without problems. Seasonal, occasional, and "binge" drinkers may be misclassified in terms of their average consumption, and what we have called non-drinkers will include occasional drinkers. Longer periods of recall and reports of average consumption are said to be less precise, so although the seven day record underestimates consumption compared with very detailed interviewer questioning about "recent occasions", its simplicity makes it the assessment of choice to many, particularly in a multi-subject, self administered questionnaire such as ours.^{43,44} In this study the possibility of biased self reporting was considered by comparing the average ranks of the ratio of reported alcohol consumption with GGT between the diagnosed CHD, undiagnosed CHD, and control groups for each sex (more extensive GGT analyses are given elsewhere⁴⁵). The three

groups were not significantly different ($p > 0.1$ for each sex), and hence there was no evidence that inaccuracies in reporting consumption had affected the alcohol *versus* CHD analyses. Lifetime non-drinkers were not identified by our questionnaire, so the "non-drinkers" groups consist of both never and ex-drinkers. Other studies^{11,12,14-16} have been able to make the distinction and found that lifetime non-drinkers were at greater risk of CHD than drinkers. While our undiagnosed CHD analysis did not include those who had given up drinking because of a positive diagnosis of CHD it might have included some ex-drinkers who had given up because of other health problems or symptoms of undiagnosed CHD.

Our results agree with most recent work, which has suggested that alcohol, at least in moderation, has a protective effect on CHD. Most of this earlier work has been concerned with mortality but our work confirms the relationship with CHD prevalence. Allowance for several coronary risk factors, some of which were not available to previous authors, has not changed the basic conclusion.

When women have been included in studies on alcohol and disease, the results have generally been similar to those for men, but at a lower dosage level, and our own findings are consistent both for risk^{1,3,8,25-28} and risk factors.⁴⁶ Women have very much lower alcohol intakes than men in Scotland but the alcohol-prevalent CHD relationship is similar to that for men, provided that the scale of measurement for alcohol is reduced.

It seems therefore that moderate alcohol consumption is both a component and a contributor to a pattern of lifestyle and risk factors associated with a reduced risk of CHD. The observation that multiple adjustment for lifestyle associations removes much of the effect, while adjustment for the measured biomedical consequences removes an additional large (and partly overlapping) component, suggests that the presumed benefit operates through both of these mechanisms. The suggestion of reduced risk in wine drinkers compared with beer is intriguing, however, and could suggest that not all alcohol sources are the same and that other components of drinks may contribute.^{23,24} On the other hand, we have previously shown that coffee consumption in the Scottish heart health study is associated with a lower prevalence of CHD than tea.⁴⁷ The reduced risk in each case associated with the newer versus the traditional beverage raises an alternative possibility, that we may have adjusted inadequately for other lifestyle associations. The spirits/beer/wine specific data are rather inadequate to present in detail here, but the tendency does not contradict current suggestions that red wine may have specific effects on coronary risk.

At a time when national policies are being framed both for preventing CHD and promoting health in general there is inevitable interest in what recommendations arise from analyses such as these and whether the so-called "safe limit" for alcohol of 21 units/week for men and 14 units/week for women should be changed.⁴⁸

In the SHHS about a fifth of all middle aged men are non-drinkers, at least over a seven day period, whereas over a third of women are non-drinkers. Yet 30.3% of men and 6.8% of women in the SHHS were exceeding the official limit. These are similar figures to the official estimate for 1986⁴⁰ and slightly higher in men and lower in women than the United Kingdom average. Alcohol is an important source of energy for men in the SHHS, with a mean percentage of energy intake from alcohol of 6.2%. Women derive a mean of only 2.5% of their energy from alcohol; indeed female alcohol consumption is very slightly negatively correlated with total energy consumption (table 3). The difference in drinking habits between the sexes is underlined by the comparison of manual and non-manual social class groups in the SHHS.³⁴ Whereas men in the manual social class drink slightly more (in percentage energy terms) than their non-manual counterparts, women in the manual social class drink considerably less ($p < 0.001$).

Current policy aims to reduce the percentage of heavy drinkers⁵⁰ but there is no policy to encourage non-drinkers to drink alcohol. The extremely high CHD rates in Scottish women,⁴² which are concentrated in precisely the manual occupational classes who have the lowest alcohol consumption, might prompt the question "Why not?" It is, however, difficult to see how one sex and occupational class group can be treated differently from the rest. Increasing average consumption would tend to increase those exceeding safe levels. CHD is only part of the disease burden of women. Large prospective studies of alcohol and mortality are increasingly reporting on other components of mortality than all causes and CHD,²⁶ and some of these suggest increased risk of breast cancer with alcohol in women. Specific advice may have to await further analyses of risks and mechanisms and greater consistency between findings, not only of the attributable benefits of alcohol consumption for CHD but also the attributable costs for other diseases, findings to which the prospective phase of the SHHS will be contributing.

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