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Alcohol consumption and risk of coronary heart disease 11

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Abstract

Objective—To investigate the hypothesis that the apparent protective effect of habitual alcohol consumption on coronary heart disease is due to drinkers at high risk of coronary heart disease becoming non-drinkers.

Design—Case-control population based study. Data were obtained from interviews with patients with non-fatal myocardial infarction and their controls and with the next of kin of those who had died of coronary heart disease and their controls.

Setting—Auckland, New Zealand.

Subjects—Two groups of cases were studied. The first comprised 227 men and 72 women with non-fatal myocardial infarction identified from a population based surveillance programme for coronary heart disease; controls were 525 men and 341 women randomly selected from the same population group and matched for age and sex. The second group comprised 128 men and 30 women who had died of coronary heart disease and had been identified from the surveillance programme; controls were a sample of the previous control group and comprised 330 men and 214 women matched for age and sex. All participants were aged 25-64 years and without diagnosed coronary heart disease.

Main outcome measures—Regular alcohol consumption; high density lipoprotein cholesterol and low density lipoprotein concentrations.

Results—Men with myocardial infarction and men who had died of coronary heart disease were more likely to have been never drinkers (had never drunk more than once a month) than controls (18% v 12% and 23% v 13% respectively). After possible confounding factors had been controlled for, people in all categories of drinking (up to more than 56 drinks per week) had at least a 40% reduction in risk of fatal and non-fatal coronary heart disease compared with never drinkers. Former drinkers also had a lower risk of non-fatal myocardial infarction than never drinkers (relative risks 0.41 and 0.10 in men and women respectively) but a similar risk of death from coronary heart disease. The reduction in risk was consistently greater in women than in men in all drinking categories but there was no clear dose-response effect in either sex.

Conclusions—The results support the hypothesis that light and moderate alcohol consumption reduces the risk of coronary heart disease. This protective effect in this population was not due to the misclassification of former drinkers with a high risk of coronary heart disease as non-drinkers.

Introduction

Epidemiological studies have consistently shown an apparent protective association between light to moderate alcohol consumption and coronary heart disease,¹ but interpreting these findings remains a subject of much debate.² On the basis of data from the

British regional heart study Shaper *et al* argue that the protective association is an artefact due to drinkers at high risk of coronary heart disease becoming non-drinkers³; classification of these former drinkers as non-drinkers would have the effect of increasing the risk of coronary heart disease in the non-drinking category and decreasing it in the drinking category. The findings of many studies which conflict with this hypothesis are questioned on methodological grounds.²

We report the findings of a large case-control study which specifically examined the association between habitual alcohol consumption and coronary heart disease. The study addresses most of the issues raised by Shaper *et al*. It was community based, included both men and women, examined non-fatal and fatal coronary heart disease, used validated measures of alcohol consumption, and differentiated former drinkers from never drinkers, as well as healthy from unhealthy people.

Methods

STUDY POPULATION

The study population included all white men and women aged 25-64 living in the Auckland statistical area between March 1986 and February 1988 who were also registered on the general electoral rolls. The electoral rolls were over 95% complete, and about 80% of the region's residents in this age group (1986 census population 420 226) were of European descent. The Auckland statistical area's population is mainly urban and accounts for about one quarter of the total population of New Zealand.

SELECTION OF CASES

Two groups of cases were included in the study (fig 1). All patients with non-fatal myocardial infarction from a World Health Organisation MONICA project register,⁴ which identifies all episodes of myocardial infarction requiring admission to hospital in the study population,⁵ were invited to participate. Similarly, the next of kin of all people who had died of coronary heart disease according to the same register, which identifies over 99% of such cases in the study population,⁶ were asked to provide information on the people who had died.

SELECTION OF CONTROLS

Controls for the subjects with myocardial infarction (group matched by age and sex) were randomly selected from the study population by using the electoral rolls as the sampling frame. Case-control ratios of about 1:1.5 in men and 1:3 in women were planned. Controls were invited to participate by letter; up to three letters were sent, followed by a telephone call to organise a visit to the study centre for interview. As information on those who had died of coronary heart disease came from their next of kin, we collected information on their controls in the same way. This

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was achieved by using the controls for the subjects with myocardial infarction, who had been interviewed at the study centre, as controls for those who had died of coronary heart disease as well and interviewing their next of kin about them. After the initial interview of the myocardial infarction controls was completed a randomly selected sample of these controls were asked if the study team could contact their next of kin, just as the next of kin of those who died of coronary heart disease had been contacted after the cases were identified. The data collected from the interviews with the next of kin of the controls were used for the case-control comparisons with those who had died of coronary heart disease. Therefore, two sets of data were collected on each control. One set, collected directly from the control, was used for case-control analyses for myocardial infarction and the other set, collected from the next of kin, was used for the case-control analyses for fatal coronary heart disease.

COLLECTION OF DATA

All subjects with myocardial infarction were interviewed in one study centre by one of three trained interviewers about three to four weeks after the infarction. Their controls were also interviewed in the same centre by the same interviewers.

A non-fasting blood sample was taken from all controls at the time of interview to estimate serum high density lipoprotein cholesterol and low density lipoprotein cholesterol concentrations. A sample was also collected from subjects with myocardial infarction as soon as possible after admission to hospital. Lipid concentrations were measured only when the sample had been taken within 24 hours of the onset of the myocardial infarction as there is little change in serum lipid concentrations in the first 24 hours after infarction.⁷ Samples were frozen and analysed blind two to four weeks after collection by a standard method.

The interviews of the next of kin for both the subjects who had died of coronary heart disease and their controls took place in the interviewee's home, usually six to eight weeks after the subject's death or the control's interview. All interviews were conducted by the same three members of staff with an identical structured questionnaire.

Regular alcohol consumption was assessed by the "typical occasions" method.⁸ Respondents were asked how often they usually drank alcohol and what they typically drank. The questions were based on usual consumption over the three months before the myocardial infarction, death, or control interview. Subjects who currently drank less than once a month were classified as former drinkers if they had ever drunk on average more than once a month and if not they were classified as never drinkers. Change in average alcohol intake in the previous 12 months was also recorded.

Participants indicated their alcohol consumption in numbers of glasses, cans, and bottles (in various sizes) of beer, spirits, liqueurs, fortified wine, and table wines. This was converted to grams of absolute alcohol by using a standard conversion table. There are about 8 g of alcohol in a typical drink (half a pint of regular beer, one glass of wine, or a single measure of spirits).¹

Cigarette smoking was assessed by the WHO MONICA project smoking questionnaire⁹; current drug treatment of hypertension was used as a proxy for hypertension; leisure time physical activity was based on regular participation in vigorous and moderate activities outside work; and socioeconomic status was assessed by using a modified version of the British registrar general's classification of social class.⁹ Prevalent coronary heart disease was assessed by the angina questionnaire of Rose *et al*.¹⁰ and questions on previous hospital admission for coronary heart disease.

VALIDATION STUDIES

High density lipoprotein cholesterol and alcohol intake—High density lipoprotein cholesterol concentration was measured in myocardial infarction controls (486 men and 299 women). There was a significant positive linear relation between reported alcohol intake and high density lipoprotein cholesterol concentration in both men and women (Spearman rank correlation coefficient 0.25, 95% confidence interval 0.16 to 0.35). Given the many factors other than alcohol which influence high density lipoprotein cholesterol concentration, this level of correlation was quite high. A similar linear trend was observed for men with myocardial infarction (n=137) (Spearman correlation coefficient 0.24, 0.07 to 0.41). There were insufficient data on women with myocardial infarction (n=38) to show any clear relation. When alcohol consumption was categorised according to numbers of drinks per week (see table IV) the mean high density lipoprotein cholesterol concentrations for each category increased in a monotonic manner with increasing alcohol consumption. Never drinkers had similar concentrations to former drinkers. The same pattern was observed for both cases and controls.

Data from controls and next of kin—Analyses based on 510 pairs of myocardial infarction controls and their next of kin showed strong agreement on frequency of drinking and amount per occasion between information given by the controls themselves and that given by their next of kin. For frequency of drinking the exact agreement of all categories (see categories used in tables II and III) was 69% and agreement within one category was 90%, while the intraclass correlation was 0.71 (95% confidence interval 0.67 to 0.75). For amount per week the equivalent indices of agreement were 57%, 92%, and 0.76 (0.72 to 0.80). The intraclass correlation is interpretable as the ratio of the within pair variation to total variation and hence approaches 1.0 as the within pair agreement improves. Under the assumption of marginal homogeneity the intraclass correlation is equal to a version of weighted κ with the weights equivalent to the square of the difference between the category scores.¹¹

ANALYSIS OF DATA

Alcohol consumption was classified into categories based on the findings of previous studies and on the distribution of the study data. All variables examined in the analyses were defined categorically. 95% Confidence intervals for differences in means and proportions between cases and controls were calculated by standard methods. Unconditional multiple logistic regression was used to calculate adjusted odds ratios as estimates of relative risks by using SAS software.¹² Potential confounders evaluated included age (in three categories), cigarette smoking (current, previous, or

Selection of cases and controls and methods of data collection

never), leisure time physical activity (regular weekly vigorous activity, moderate activity, or neither), current treatment for hypertension (yes or no), socioeconomic status (high, middle, or low), and change in alcohol consumption in the previous 12 months (more, less, or no change).

Results

PARTICIPANTS

A total of 2301 questionnaires were completed relating to 1720 people (a questionnaire was completed by the next of kin of 581 controls). Response rates in the patients with myocardial infarction were 90% (n=359) in men and 76% (n=122) in women; for myocardial infarction controls they were 80% (n=561) and 85% (n=379); for people who had died of coronary heart disease 83% (n=249) and 79% (n=50); and for controls for those who had died of coronary heart disease 85% (n=355) and 79% (n=226). Cases and controls with prevalent coronary heart disease were excluded from all subsequent analyses because of the concern that those with symptoms might change their drinking habits. This exclusion had little effect on the reported findings, however. People with prevalent coronary heart disease accounted for 36% of men and 41% of women with myocardial infarction, 6% of male and 10% of female myocardial infarction controls, 49% of men and 40% of women who had died of coronary heart disease, and 7% of male and 5% of female coronary heart disease controls.

After participants with prevalent coronary heart disease had been excluded 1165 people were available for the case-control analysis for myocardial infarction and 702 people for the coronary heart disease analyses. Table I shows the distribution of the major risk factors for coronary heart disease and other characteristics for cases and controls. As expected, there were striking differences in the prevalence of smoking and the treatment of hypertension between cases and controls for both myocardial infarction and fatal coronary heart disease. Controls in each category were also more likely to participate in vigorous or moderate leisure time physical activity than cases. The mean ages were similar within each case-control category except for men who had died of coronary heart disease; all analyses were nevertheless stratified by age group. The social class distributions (not shown) were also similar.

FREQUENCY OF DRINKING

Table II shows the frequency of drinking in cases and controls by sex. Men with myocardial infarction and those who had died of coronary heart disease were more likely to have been never drinkers than controls

TABLE II—Frequency of drinking in cases and controls. Figures are percentages (numbers) of people unless otherwise stated

Drinking frequency	Cases with myocardial infarction	Controls	Cases who had died of coronary heart disease	Controls
<i>Men</i>				
No of men	227	525	128	330
Never drinkers*	18 (41)	12 (63)	23 (29)	13 (43)
Former drinkers	6 (14)	6 (32)	8 (10)	3 (10)
<Once/day	43 (98)	49 (257)	33 (42)	51 (168)
Once/day	31 (70)	30 (158)	31 (40)	32 (106)
>Once/day	2 (5)	3 (16)	5 (6)	1 (3)
<i>Women</i>				
No of women	72	341	30	214
Never drinkers*	45 (32)	24 (82)	70 (21)	23 (49)
Former drinkers	3 (2)	5 (17)	13 (4)	3 (6)
<Once/day	37 (27)	47 (160)	7 (2)	49 (105)
Once/day	15 (11)	22 (75)	10 (3)	24 (51)
>Once/day		2 (7)		1 (2)

*Includes those who drink <once/month.

TABLE III—Weekly alcohol consumption in regular drinkers. Figures are percentages (numbers) of people unless otherwise stated

Alcohol consumption (drinks/week)	Cases with myocardial infarction	Controls	Cases who had died of coronary heart disease	Controls
<i>Men</i>				
No of men	173	430	88	277
≤4	24 (42)	31 (133)	32 (28)	40 (111)
5-14	29 (50)	32 (138)	26 (23)	28 (78)
15-35	25 (43)	24 (103)	21 (18)	20 (55)
36-56	10 (17)	8 (34)	7 (6)	7 (19)
>56	12 (21)	5 (21)	14 (12)	5 (14)
<i>Women</i>				
No of women	37	242	5	158
≤4	66 (24)	54 (131)	40 (2)	52 (82)
5-14	16 (6)	30 (73)	20 (1)	30 (47)
15-35	10 (4)	13 (31)	20 (1)	14 (22)
>35	8 (3)	3 (7)	20 (1)	4 (6)

(the never drinking category includes those who had never drunk more than once a month). The proportion of former drinkers was identical for patients with myocardial infarction and controls whereas almost three times as many of those who had died of coronary heart disease as controls had been former drinkers. Male controls were more likely to be light drinkers (less than once a day) than were cases, although the proportions in the once a day category were similar. Few men drank more than once a day.

The pattern of drinking in women differed from that in men, principally because of the larger proportion of never drinkers, particularly among cases. Almost half of those with myocardial infarction and more than two thirds of those who had died of coronary heart disease were never drinkers compared with about one quarter of the controls. In addition, more than four times as many of those who had died of coronary heart disease as controls were former drinkers. As a result of the concentration of cases in the non-drinking categories there was a greater proportion of controls in all current drinking categories.

Table III shows usual alcohol consumption per week among cases and controls who were regular drinkers by sex. In men there was a tendency for more cases than controls to be in the heavier drinking categories, whereas in women there was no clear trend in the myocardial infarction case-control category. The small number of women who had died of coronary heart disease who were drinkers precluded any detailed comparisons with controls.

EFFECT OF ALCOHOL ON RISK OF CORONARY HEART DISEASE

Table IV gives the relative risks of myocardial infarction and fatal coronary heart disease associated with habitual alcohol consumption for cases and controls by sex. The reference category is never

TABLE I—Major characteristics of cases and controls. Figures are percentages (numbers) of people unless otherwise stated

	Myocardial infarction			Fatal coronary heart disease		
	Cases	Controls	Cases v controls	Cases	Controls	Cases v controls
<i>Men</i>						
No of men	227	525		128	330	
Mean age (years)	51.5	50.9	0.6	55.9	51.3	4.6
Leisure time physical exercise:						
Vigorous	15 (34)	23 (121)	-8	12 (15)	21 (69)	-9
Moderate	37 (84)	38 (200)	-1	25 (32)	31 (102)	-6
Current smokers	48 (109)	21 (110)	27	50 (64)	21 (69)	29
Hypertension*	18 (41)	10 (53)	8	28 (36)	10 (33)	18
<i>Women</i>						
No of women	72	341		30	214	
Mean age (years)	55.4	53.9	1.5	56.6	54.8	1.8
Leisure time physical exercise:						
Vigorous	10 (7)	14 (48)	-4	3 (1)	15 (32)	-12
Moderate	25 (18)	43 (147)	-18	17 (5)	31 (66)	-14
Current smokers	58 (42)	23 (78)	35	40 (12)	21 (45)	19
Hypertension*	32 (23)	16 (55)	16	40 (12)	16 (34)	24

*Subjects receiving current drug treatment for hypertension.

TABLE IV—Relative risk* (95% confidence intervals) of non-fatal myocardial infarction and deaths from coronary heart disease associated with habitual alcohol consumption

Alcohol consumption (drinks/week)	Men		Women	
	Myocardial infarction	Fatal coronary heart disease	Myocardial infarction	Fatal coronary heart disease
Never drinkers	1.00	1.00	1.00	1.00
Former drinkers	0.41 (0.18 to 0.94)	1.10 (0.30 to 3.31)	0.10 (0.02 to 0.54)	1.10 (0.20 to 4.2)
≥4	0.57 (0.33 to 1.01)	0.39 (0.19 to 0.79)	0.51 (0.25 to 1.03)	0.04 (0.01 to 0.23)
5-14	0.57 (0.32 to 1.00)	0.38 (0.17 to 0.83)	0.17 (0.06 to 0.49)	0.02 (0.001 to 0.30)
15-35	0.51 (0.29 to 0.92)	0.37 (0.16 to 0.84)	0.15 (0.04 to 0.54)	0.47 (0.003 to 0.70)
36-56	0.58 (0.27 to 1.28)	0.18 (0.05 to 0.61)	0.42 (0.04 to 4.4)	0.86 (0.02 to 32.30)
>56	1.23 (0.36 to 1.82)	0.54 (0.19 to 1.54)	—	—

*Calculated using logistic regression and adjusted for age, smoking (current and previous), hypertension, social class, exercise (vigorous and moderate), and recent change in drinking.

drinkers and the risk estimates are adjusted for age, smoking, treatment for hypertension, exercise, social class, and change in alcohol consumption in the previous year. In both men and women former drinkers had a reduced risk of myocardial infarction compared with never drinkers but an identical risk of fatal coronary heart disease. In all current drinking categories up to 56 drinks a week drinking seemed to be associated with a reduced risk of both myocardial infarction and fatal coronary heart disease in men and women. Given the small numbers of participants in the higher drinking categories, the 95% confidence intervals for these point estimates were wide. In general the reduction in risk associated with alcohol consumption was greater in women than in men. There was no obvious dose-response effect. The only drinking categories showing a relative risk point estimate above 1.0 were risk of myocardial infarction in men drinking more than 56 drinks a week and risk of fatal coronary heart disease in former drinkers; in each of these cases the point estimates were close to 1.0.

Of the potential confounders examined only cigarette smoking had a consistent effect on the relative risks. The protective association between alcohol and coronary heart disease was strengthened after adjusting for cigarette smoking because of the strong associations between smoking and drinking and between smoking and coronary heart disease. The unadjusted relative risks were in general 10-20% higher than adjusted risks; the pattern, however, was identical.

FURTHER ANALYSES

Further analyses (not shown) restricted to the subgroup of people without current medical problems—that is, excluding those receiving treatment for hypertension, other circulatory problems, renal disease, or gastrointestinal disease—showed the same pattern as in table IV.

Relative risk estimates of myocardial infarction in those participants with lipid data were calculated with and without adjustment for high density lipoprotein cholesterol and low density lipoprotein cholesterol concentrations (not shown). Because of the reduced numbers the only other variables adjusted for were age and smoking. Controlling for low density lipoprotein cholesterol concentration had no apparent effect on the risk estimates but controlling for high density lipoprotein cholesterol weakened the association, particularly in men and in the heavier drinking categories.

We examined the effects of wine, beer, and spirits separately and found no differences in effect by alcohol type, but the power of the study to differentiate between beverage types was low.

Discussion

The findings of this study are consistent with numerous others reporting an apparent protective

association between habitual drinking and non-fatal myocardial infarction in men.^{1 13-16} For women there is less information, but our findings are consistent with the available data, which show a similar pattern to that in men.¹⁷ In our study the apparent protective effect of habitual alcohol consumption was also similar for non-fatal and fatal coronary heart disease. In addition, our study provides no support for the theory that the observed association is due to drinkers with a high risk of coronary heart disease reducing or stopping drinking.² Former drinkers had a similar risk of dying from coronary heart disease to people who never drank more than once a month, and a lower risk of non-fatal myocardial infarction. Moreover, the findings were similar whether or not participants with prevalent coronary heart disease were included.

BIAS

Our observations are unlikely to be due to selection bias, given the completeness of the disease register used to identify cases, a sampling frame which included all individuals in the study population, and the relatively high response rates for both cases and controls. Information bias is of more concern because the data collection was carried out retrospectively. There was, however, little public awareness of the relation between alcohol and coronary heart disease at the time of the study, and the interviewers were not informed of the study hypotheses. Moreover, the data were collected by using a highly structured questionnaire in an identical setting for cases and controls. To reduce the opportunity for bias due to the effect of pre-morbid conditions or symptoms on drinking alcohol as suggested by Shaper *et al*² the analyses excluded all study participants with prevalent coronary heart disease. In subsequent analyses further exclusions of subgroups with other morbidity were made, and this had little effect on the overall findings. In addition, as drinking behaviour can change over relatively short time periods, the case-control study may be the most appropriate study design to investigate associations between alcohol and disease as it is probably less prone to misclassification of exposure than follow up studies.

ACCURACY OF DATA

A major criticism of the published work on alcohol and coronary heart disease is the lack of validation of alcohol consumption.² The accuracy of the self reported alcohol consumption data on which our paper is based is supported by the two validation studies. Firstly, we found a significant linear relation between self reported intake and high density lipoprotein cholesterol concentration, which has been well described.^{18 19} Secondly, the consistency in reporting of intake from individuals and their next of kin suggests that our data were reasonably accurate.

CLASSIFICATION OF NON-DRINKERS

The most valid criticism of much of the published work on alcohol and coronary heart disease is the loose designation of non-drinkers. By differentiating between former drinkers and people who had never drunk regularly we were able to examine the hypothesis that the apparent protective effect of drinking is related to drinkers at increased risk of coronary heart disease stopping drinking and being classified as non-drinkers, having the effect of increasing the apparent risk of coronary heart disease in non-drinkers and decreasing the risk in drinkers. We defined never drinkers as lifetime teetotallers and those who had never drunk alcohol more than once a month, because alcohol drunk less than once a month is unlikely to have any biological effects. Little difference in risk of coronary heart disease has been found between abstainers and very infrequent drinkers.²⁰

In our study former drinkers actually had a lower risk of myocardial infarction than never drinkers and a similar risk of fatal coronary heart disease. This strongly suggests that in our study population the migration of drinkers into the non-drinking group is not the explanation for the reduced risk of coronary heart disease in drinkers. The only major difference we found between never drinkers and former drinkers was a higher prevalence of cigarette smoking in the former drinkers. Some former drinkers may have described themselves as never drinkers, but we believe this is unlikely to have accounted for our results for several reasons. Firstly, the findings based on data from a proxy source (next of kin) also showed no increase in risk among former drinkers, and relatives are probably less likely to provide deliberately false data than the primary respondents. Secondly, there would have had to have been a large degree of misclassification, particularly in women, to account for the findings. Given that the self reported prevalence of never drinking in women is double that in men, dilution of the never drinking category with former drinkers at high risk of coronary heart disease should have a more pronounced effect in men than women; the opposite was in fact observed. Therefore there is no evidence to suggest that the observed effect of alcohol on risk of coronary heart disease is an artefact due to confounding by comorbidity.

Klatsky *et al* examined people admitted to hospital for coronary heart disease in the Kaiser Permanente study and found that never and former drinkers had a similar risk of coronary heart disease.²⁰ Both groups were at higher risk than current drinkers. Similar findings were reported in the Honolulu heart study.²¹ The British regional heart study findings suggest that migration of drinkers at high risk of coronary heart disease to non-drinking groups could account for the apparent protective effect of drinking.³ However, migration would have to have occurred before the baseline exposure measurement, yet this cannot be determined from the data.²² Moreover, if alcohol was protective, migration after baseline measurement would tend to obscure the protective effect.²² An additional concern about the British regional heart study's participants is the unusually high proportion of former drinkers in the non-drinking category. Over 70% of non-drinkers were former drinkers whereas in our study and many others the equivalent figure was about 25%.^{20 21}

NON-DRINKERS AS REFERENCE GROUP

Marmot suggested that teetotallers may not be the appropriate reference group when examining the possible risks of alcohol because they are a small, unusual group and may have an increased risk of coronary heart disease for other reasons—for example, personality type or diet.¹ However, almost a quarter of the female controls in our study were never drinkers, indicating that female non-drinkers are not uncommon yet still seem to have a greater risk of coronary heart disease than current drinkers. Other studies in which a third or more of the participants were never drinkers also show a protective effect of light and moderate drinking.^{20 21} In addition, the consistency of findings in many populations in different countries with wide variations in drinking patterns suggests that an unknown confounder is an unlikely explanation for the findings.¹

POSSIBLE REASONS FOR PROTECTIVE EFFECT

As shown in several previous studies there was a positive association between high density lipoprotein cholesterol concentration and alcohol consumption.^{18 19} However, high density lipoprotein cholesterol concentration could explain only part of the protective effect

of alcohol.¹⁹ Alcohol is known to influence platelet aggregation²³ and fibrinogen concentration,²⁴ which might account in part for the protective effect observed. The lack of any dose-response relation could be due to greater inaccuracy of self reported drinking in the higher drinking categories. It is also feasible that alcohol has both positive and negative effects on the risk of coronary heart disease which act simultaneously. The effect of alcohol on blood pressure²⁵ could negate any further increase in a protective effect associated, for example, with increasing high density lipoprotein cholesterol concentration at higher levels of consumption.

CONCLUSIONS

In summary, this study provides support for the hypothesis that light or moderate alcohol consumption reduces the risk of both fatal and non-fatal coronary heart disease in men and women. As our study included few very heavy drinkers we could not assess the risk of coronary heart disease associated with high levels of consumption. We have, however, answered most of the criticisms raised about previous studies showing a protective association between alcohol consumption and coronary heart disease. The data on alcohol consumption were validated and the study was population based. Most importantly, we were able to differentiate former drinkers from never drinkers, and there is no evidence from our study that the protective association observed between alcohol consumption and coronary heart disease is due to the migration of former drinkers to non-drinking groups. Finally, this study examined only coronary heart disease end points, and the public health implications of possible beneficial effects of alcohol consumption on coronary heart disease must be balanced against the burden of other morbidity and mortality caused by heavy alcohol intake.

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Prevalence of primary fibromyalgia in the Finnish population

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Abstract

Objective—To obtain descriptive epidemiological data on fibromyalgia and its components in a representative sample of the Finnish population.

Design—Cross sectional study of 8000 Finns aged 30 or more invited for screening and a main examination for musculoskeletal disorders and other major disorders.

Setting—A mobile clinic.

Population—7217 subjects who attended the screening phase; 3434 subjects positive on screening who attended the main examination for musculoskeletal disorders.

Main outcome measures—Musculoskeletal, mental, and other symptoms detected by interview and questionnaire; results of standardised clinical examination of the musculoskeletal system; operational definition of fibromyalgia; mortality at 10 years.

Results—The prevalence of fibromyalgia was low (54 cases; 0.75%) and related to age (peak prevalence at 55-64 years), female sex (twice as prevalent in women), occupation (no cases among 1596 white collar professionals), level of education (strong inverse gradient), and high levels of physical stress at work. No significant associations were found with body mass index, smoking, or mental stress at work. The prevalence of fibromyalgia was sensitive to even minor modifications of the definition. Fibromyalgia was strongly coincident with many other disorders, especially musculoskeletal conditions. Fibromyalgia did not predict mortality.

Conclusion—Descriptive epidemiological data offer little support for the concept of fibromyalgia.

Introduction

Fibromyalgia (sometimes called primary fibromyalgia or fibrositis) has been referred to as an independent disease entity or certainly a syndrome.¹ It has been claimed to be highly prevalent and frequently misdiagnosed.^{2,3} Clinical descriptions give the impression that it is often severely disabling,^{4,5} but most samples are from referral centres and thus subject to a strong selection bias. To our knowledge no one has examined the prevalence of primary fibromyalgia in the general population.

The Social Insurance Institution in Finland has conducted a mobile clinic survey of the health status of a representative sample of the Finnish population aged 30 and over—the mini-Finland health survey.⁶ In this paper we use these data to report the distribution and mutual correlations of the components of the primary fibromyalgia syndrome in the Finnish population. Our principal aims were to estimate the prevalence of fibromyalgia and detect determinants of its occurrence.

Population and methods

The data were collected in 1977-80 as part of the mini-Finland health survey, designed to provide infor-

mation about the population's health, its need for care and rehabilitation, the consequences of disease, and factors affecting health.⁶ The study population was a two stage cluster sample⁷ of 8000 people (3637 men, 4363 women) who were representative of all Finnish adults aged 30 years or more. In the first stage 40 regions (mostly single municipalities, of which there are 461 in Finland) were selected to represent the population of Finland with regard to geographical area, degree of urbanisation, and proportion of people employed in industry and agriculture. In the second stage a sample of inhabitants was drawn systematically from the population register of each region.

The examinations were carried out by the mobile clinic of the Social Insurance Institution in two main phases, a screening phase and a main examination phase. A total of 7217 subjects (90.2% of the sample) participated in the screening phase. The distributions of sex, age, marital status, and level of education among the participants corresponded closely with those in the whole Finnish population.⁶

FIELD SURVEY

The screening protocol included several interview items that have been defined as components of the primary fibromyalgia syndrome¹—namely, reported "hurt all over," enumeration of painful body sites, general fatigue, poor sleep, anxiety and tension, and gastrointestinal problems. The irritable bowel syndrome was not sought specifically. Moderate or severe levels of the symptoms were considered relevant in this study to detect fibromyalgia.

Subjects (n=3775; 52.3%) who in the screening phase reported any moderate or severe musculoskeletal symptoms or had impaired function of at least one joint were invited to a clinical examination to evaluate musculoskeletal morbidity; 3434 (91.0%) attended.⁶ They were examined by one of the mobile clinic physicians, and all conditions that satisfied pre-established criteria for major musculoskeletal disorders were independently recorded. Although this examination was initially not designed to detect primary fibromyalgia, it included a systematic recording of pressure tenderness in the wrists, elbows, trapezoid muscles, shoulder joints, knee joint region, and Achilles tendons. These represent many but not all of the points suggested in previous studies of fibromyalgia.

Other illnesses were also screened for and were investigated separately by similar diagnostic procedures. Mental disorders were sought by the general health questionnaire^{8,9} and diagnosed by the present state examination and a computerised coding algorithm (CATEGO-ID).¹⁰

Deaths among the study population were traced to the end of 1989 by linkage with the Finnish population register. Ascertainment was complete.

DIAGNOSIS OF FIBROMYALGIA

For most analyses we used an operational definition of fibromyalgia adapted from Yunus *et al*.¹ This

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