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Original article

Coffee and wine consumption is associated with reduced mortality from alcoholic liver disease: follow-up of 219,279 Norwegian men and women aged 30–67 years

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ABSTRACT

Purpose: To study the association between coffee and alcoholic beverage consumption and alcoholic liver disease mortality.
Methods: In total, 219,279 men and women aged 30–67 years attended cardiovascular screening in Norway from 1994 to 2003. Linkage to the Cause of Death Registry identified 93 deaths from alcoholic liver disease. Coffee consumption was categorized into four levels: 0, 1–4, 5–8, and greater than or equal to 9 cups/d and alcohol consumption as 0, greater than 0 to less than 1.0, 1.0 to less than 2.0, and greater than or equal to 2.0 units/d, for beer, wine, liquor, and total alcohol consumption.
Results: The hazard ratios per one category of consumption were 2.06 (95% confidence interval 1.62–2.61), 0.68 (0.46–1.00), and 2.54 (1.92–3.36) for beer, wine, and liquor, respectively. Stratification at 5 cups/d (the mean) revealed a stronger association between alcohol consumption and alcoholic liver disease at less than 5 versus 5 or more cups/d. With less than 5 cups/d, 0 alcohol units/d as reference, the hazard ratio reached to 25.5 (9.2–70.5) for greater than or equal to 2 units/d, whereas with greater than or equal to 5 cups/d, it reached 5.8 (1.9–17.9) for greater than or equal to 2 units/d. A test for interaction was significant ($P = .01$).
Conclusions: Coffee and wine consumption were inversely associated with alcoholic liver disease death. Total alcohol consumption was adversely associated with alcoholic liver disease mortality and the strength of the association varied with the level of coffee consumption.

Introduction

Alcoholic liver disease (ALD) is the leading cause of death from excessive alcohol consumption among Western European adults [1]. Norway has the lowest mortality from liver cirrhosis among Western European countries [2]. There is a well-known association between total alcohol consumption and the risk of ALD [3–6]. However, the type of alcoholic beverages may be important for the strength of this association. Two Danish studies found that wine drinkers were at lower risk for liver cirrhosis than consumers of beer and liquor [5,7]. Further protection may be derived from coffee consumption, as a number of studies have reported an inverse association between coffee consumption and the risk of liver cirrhosis [8–11] and two meta-analyses conclude that coffee consumption may reduce the risk of hepatic fibrosis and cirrhosis [12,13]. We have previously shown that coffee consumption is inversely associated with mortality from liver cirrhosis [14], but alcohol consumption in that study was crudely measured (as yes or no alcohol during a week) and without distinguishing between different alcoholic beverages [14]. In the present study, we have examined the association of coffee and alcohol consumption, both separately and in combination, with ALD mortality, taking type of alcoholic beverage and amount into consideration.

The authors have no conflicts of interest to disclose.

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Material and methods

In 1994–1999, a health screening (known as the Age-40 programme) was carried out in 15 counties of 19 Norwegian counties. All men and women aged 40–42 years were invited in addition to a random sample aged 43–44 years. The screening consisted of filling in a questionnaire and a simple physical examination. The questionnaire encompassed questions on a number of dietary habits including coffee drinking and alcohol consumption, as well as other life-style and health-related topics such as smoking habits, physical activity during leisure, history of cardiovascular disease and diabetes, education level, disability pension, and sick leave or rehabilitation wages. The screening examination comprised anthropometric and blood pressure measurements. Height and weight were measured to the nearest centimeter and half kilogram, respectively, whereas blood pressure and pulse were measured three times using an automatic device (Dinamap), with the subject in a sitting position. A nonfasting blood sample was drawn and serum analyzed for total cholesterol and triglycerides [15,16]. In addition to this Age-40 programme, we have used data from the CONOR study from the same period and with the same protocol [17]. The CONOR study included men and women aged 20 years or older. We restricted included CONOR study sample to those aged 30–67 years. All eligible persons received an invitation letter with the questionnaire printed on the reverse side. Enclosed with the letter was a printout giving alternative places and times of attendance [15]. All invited persons were asked to fill in the questionnaire at home and bring it to the place of examination, where a nurse checked it, and omissions and logical inconsistencies were corrected according to a written protocol.

Study population

A total of 222,656 attended the screening. The participation was 69% in the Age-40 programme and 62% in the CONOR study. We excluded 3377 persons who did not answer the question on alcohol or coffee. The study population comprised 219,279 participants. We did not know if the participants had ALD when attending the screening and these participants, if any, are included.

Exposure variables

The questions on coffee consumption read (1) How many cups of boiled coffee do you drink daily? (2) How many cups of other types of coffee do you drink daily? The participants filled in the number of cups. Boiled coffee (a term directly translated from the Norwegian questionnaire) also known as camp coffee, pour over coffee or drip-brewed coffee, is any type of coffee brewed without a paper filter between coffee grounds and water. No attempt was made to standardize cup size. We defined categories 0, 1–4, 5–8, and greater than or equal to 9 cups/d. These were the preset categories in the questionnaire used before 1994 [18]. The questions on alcohol were (1) Are you a teetotaler? (yes, no), (2) How many times per month do you usually drink alcohol? (*Do not include low-alcohol beer. Write 0 if less than 1 time per month*), (3) How many glasses of beer, wine, or liquor do you usually drink during 2 weeks? (*Do not include low-alcohol beer. Write 0 if you don't drink alcohol*). The participants estimated their typical number of glasses separately for beer, wine, and liquor for a 2-week period. Missing number of glasses was recoded to zero if the participants had answered alcohol question 2 or had answered that they were teetotalers. A glass of beer was assumed to be 500 mL at 4.6% alcohol by volume. The corresponding figure for a glass of wine or a shot of liquor was 175 mL at 12% and 40 mL at 40%, respectively. Total units of alcohol were defined as (a serving of beer was calculated as 500 ml $\times$ 4.6 alcohol volume [AV] + wine as 175 ml $\times$ 12% AV + liquor as 40 ml $\times$ 40% AV/14 d $\times$ 1000).

Previous history of chronic disease

A group with self-reported ill health included 37,686 participants who reported a history of cardiovascular disease, diabetes, asthma, or cancer or reported to receive disability pension or sickness benefit. A history of cancer at baseline was confirmed by linkage to the Cancer Registry of Norway. Altogether, this amounted to 37,686 persons. The information about ill health was included as a covariate in the multivariate analyses, in addition to coffee and alcohol, sex, age, total cholesterol, triglycerides, smoking, body mass index, systolic blood pressure, and education.

Follow-up and endpoints

The participants accrued person-years from date of examination to date of death, date of emigration, or 31 December 2013, whichever came first. ALD was defined as ICD-9571.0-571.3 or ICD-10 K70. Altogether, 93 persons died from ALD during follow-up and 91 remained eligible for the multivariate analysis due to missing covariate information.

Statistics

We used the direct method to adjust for sex and age (5-year age groups) [19]. The method produces a weighted average of the stratum-specific rates. We used Cox proportional hazards regression when accounting for several potential confounders. Number of units/d of alcohol and number cups of coffee/d were defined as dummy variables with zero units/d of alcohol and zero cups of coffee/d as the reference categories, respectively. When estimating trend, the two variables were applied as continuous variables in the model with values 1, 2, 3, and so forth according to the number of categories in the table. Cox models with and without an interaction term were used to assess risk-ratio modification. Spline curves were graphed with the original number of alcohol units and number of coffee cups/d, using the mkspline package with restricted cubic spline construction (4 knots) combined with the xblc package in STATA [19].

Ethics

The study was approved by the Regional Committees for Medical and Health Research Ethics and The Norwegian Data Protection Authority.

Results

Characteristics by coffee consumption

Ten percent of the study population did not drink coffee and 13 percent drank 10 or more cups per day (Table 1). The majority in the highest consumption group were male. Total alcohol consumption increased with increasing cups of coffee/d. Smoking prevalence increased by 51 percentage points from lowest to highest coffee category. The two extreme categories of coffee consumption: 0 and 9 or more cups/d, respectively, had the highest prevalence of self-reported ill health. As some people have used both types of coffee and all three types of alcohol, some sums will exceed 100%.

Table 1
Baseline characteristics by coffee consumption adjusted for sex and age

Variable	Cups of coffee/d			
	0	1–4	5–8	≥9
N	21,764	82,728	84,077	30,710
Percentages (of N)				
Male	44	42	48	62
Wine	32.8	48.6	43.1	32.1
Beer	32.2	45.2	47.2	47.3
Liquor	17.0	25.7	32.4	39.0
Smoking	17.4	22.2	44.1	67.2
Ill health	18.2	16.5	16.7	19.8
Means				
Age (y)	42	43	44	43
Cups of coffee/d	0	3.0	6.0	11.4
Alcohol (units/d)	0.47	0.63	0.68	0.74
Total cholesterol (mmol/L)	5.4	5.6	5.7	5.9
Triglycerides (mmol/L)	1.8	1.7	1.7	1.8
Systolic blood pressure (mm Hg)	127	129	130	130
Body mass index (kg/m^2)	25.9	25.5	25.7	25.9
Hard physical activity* (medians)	2	2	2	2
Education† (medians)	2	2	2	2

Men and women aged 30–67 years.
* None = 1, <1 h/wk = 2, 1–2 h/wk = 3, ≥3 h/wk = 4.
† ≤10 y = 1, 11–12 y = 2, 13 y = 3, 14–16 y = 4, ≥17 y = 5.

Characteristics by alcohol consumption

Almost one-third reported zero units of alcohol/d and 7% reported two or more units/d (Table 2). The proportion of males increased with each increasing consumption category. The smoking prevalence, as well as the level of total cholesterol, triglycerides, systolic blood pressure, hard physical activity, and educational attainment increased with increasing number of alcohol units/d. The prevalence of ill health was highest in the nonconsumer group.

Absolute mortality by coffee and alcohol consumption

Table 3 shows a distinctly higher mortality from ALD among drinkers of 2 or more units/d compared to the three lower consumption groups. The pattern is similar within the coffee consumption groups with a varying strength of the association. Mortality decreased with number of coffee cups/d with an upturn at greater than or equal to 9 cups/d. The association between cups/d and ALD mortality points in the opposite direction for alcohol abstainers.

Hazard ratios by coffee consumption and by alcohol consumption

The spline curves showed hazard ratios below 1.0 with a leveling off or turning point at 7–8 cups of coffee/d (Fig. 1). The upper confidence limit stayed below 1.0 at more than three cups/d. The hazard ratios for alcohol consumption were largely above 1.0 and with a strong increase from 0.3 alcohol units/d. Note that the Y-axis is on the log scale and is different for coffee and alcohol.

Hazard ratios by coffee and alcohol consumption

With a cutoff of 5 coffee cups/d, the mean number of cups in this study population, the association between alcohol consumption

Table 2
Baseline characteristics by alcohol consumption adjusted for sex and age

Variable	Alcohol units/d			
	0	>0–<1	1–<2	≥2
N	71,230	98,612	35,268	14,169
Percentages (of N)				
Male	34.9	45.4	64.2	81.2
Wine	0	58.7	71.2	73.0
Beer	0	58.1	80.6	90.7
Liquor	0	36.0	55.6	64.3
Smoking	32.0	36.1	41.8	47.3
Ill health	22.3	15.3	13.2	14.6
Means				
Age (y)	44	43	43	43
Cups of coffee/d	4.8	5.0	5.3	5.7
Alcohol (units/d)	0	0.50	1.40	3.08
Total cholesterol (mmol/L)	5.7	5.6	5.7	5.8
Triglycerides (mmol/L)	1.7	1.7	1.8	2.0
Systolic blood pressure (mm Hg)	130	129	130	132
Body mass index (kg/m^2)	26.0	25.4	25.6	25.9
Hard physical activity* (medians)	2	2	2	2
Education† (medians)	2	2	2	2

Men and women aged 30–67 years.
* None = 1, <1 h/wk = 2, 1–2 h/wk = 3, ≥3 h/wk = 4.
† ≤10 y = 1, 11–12 y = 2, 13 y = 3, 14–16 y = 4, ≥17 y = 5.

Table 3
Mortality from alcoholic liver disease by coffee and alcohol consumption

Units/d	Cups of coffee/d				Total
	0	1–4	5–8	≥9	
0					
Person-years	168167	404932	417514	142692	1133305
Deaths	0	5	6	4	15
Per 100,000 person-years*	0	1.4	1.5	3.0	1.4
>0–<1.0					
Person-years	121317	629141	633987	190861	1575306
Deaths	5	8	10	6	29
Per 100,000 person-years*	6.4	1.3	1.6	2.9	1.9
1.0–<2.0					
Person-years	35,636	203600	236879	79,565	555680
Deaths	1	9	5	3	18
Per 100,000 person-years*	3.2	4.6	2.1	2.4	1.6
≥2.0					
Person-years	15,763	72,679	92,479	38,522	219443
Deaths	6	15	6	4	31
Per 100,000 person-years*	31.3	19.1	6.3	20.4	16.6
Total					
Person-years	340883	1310352	1380859	451640	3483734
Deaths	12	37	27	17	93
Per 100,000 person-years*	4.4	2.9	1.9	3.5	2.7

Sex and age-adjusted rates per 100,000 person-years.
Men and women aged 30–67 years.
* Adjusted for sex and age.

and ALD mortality was steeper below 5 cups/d than for 5 or more cups/d (Table 4). A test for interaction gave $P = .01$.

Hazard ratios by type of alcohol

Adjusted hazard ratios for different alcohol types as well as coffee intake are presented in Table 5. Mortality from ALD increased with increasing beer and liquor consumption, whereas it decreased with increasing wine consumption. The confidence interval for wine consumption did not overlap the confidence intervals for the two other alcohol types. Coffee cups/d was inversely associated with ALD at all levels of alcohol consumption.

Sensitivity analyses

We did two sensitivity analyses; one where we excluded the first 3 years of follow-up and one where we excluded those with a diagnosis of cancer at baseline. The overall results remained unchanged in both instances.

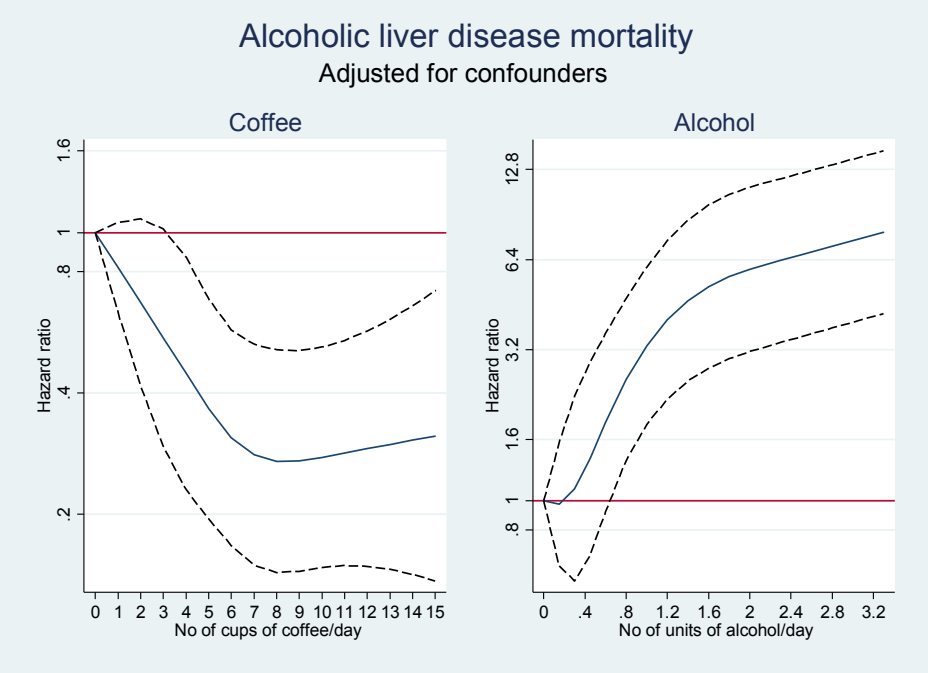

Fig. 1. Adjusted hazard ratios, with 95% confidence band, for alcoholic liver disease mortality by number of coffee cups/d and number of alcohol units/d. Men and women aged 30–67 years. The Y-axes are on the log scale and they are different for coffee and alcohol. The reference groups are zero coffee cups/d and zero alcohol units/d. Blue lines represents the adjusted hazard ratios. Dashed lines are the upper and lower 95% confidence band. Red lines are the hazard ratios equal to 1, that is no association.

Table 4
Number of deaths (N) of alcoholic liver disease and hazard ratios with 95% confidence intervals

Coffee, Cups/d	Alcohol, Units/d	N deaths	HR* (95% CI)	HR† (95% CI)
<5	0	5	Ref	Ref
<5	>0–<1.0	13	2.12 (0.75–5.95)	2.25 (0.80–6.37)
<5	>1.0–<2.0	10	5.10 (1.73–15.1)	5.20 (1.74–15.5)
<5	≥2.0	21	28.2 (10.4–76.6)	25.5 (9.20–70.5)
≥5	0	10 (9)‡	1.93 (0.66–5.67)	1.02 (0.34–3.09)
≥5	>0–<1.0	16 (15)‡	2.26 (0.82–6.20)	1.41 (0.50–3.96)
≥5	>1.0–<2.0	8	3.01 (0.97–9.35)	2.04 (0.65–6.47)
≥5	≥2.0	10	9.01 (3.00–27.1)	5.80 (1.88–17.9)

Men and women aged 30–67 years.
* Adjusted for sex and age.
† Adjusted for sex, age, total cholesterol, triglycerides, smoking, body mass index, systolic blood pressure, education, and ill health.
‡ N deaths in†.

Discussion

We found an adverse association between alcohol consumption and mortality from ALD and an inverse association between coffee consumption and mortality from ALD. There was a risk-ratio modification between the two exposures on the outcome; the association between alcohol consumption and liver cirrhosis mortality was weaker at higher levels of coffee consumption. When considering type of alcohol consumed, beer and liquor consumption were adversely and wine consumption was inversely associated with ALD mortality.

Several hypotheses have been put forward to explain the favorable effect of coffee on liver cirrhosis. Dranoff et al has reviewed some of these [20]. They emphasized the antifibrotic effect of caffeine. Some studies have on the other hand found an inverse association also for decaffeinated coffee [9,21], suggesting another compound than caffeine is implicated. Furthermore, Goh et al found no association between daily caffeine intake and nonviral hepatitis-related cirrhosis after adjustment for coffee consumption in an Asian population [11]. Shi et al reported from studies on rats that anti-inflammatory action of chlorogenic acid is one of the mechanisms to improve liver cirrhosis [22]. In a study of healthy men, coffee consumption attenuated hepatic insulin resistance but did not decrease intrahepatocellular lipids induced by fructose overfeeding [23]. We can only speculate about which substances in coffee that might have a favorable effect.

Our finding of a favorable association for wine drinking as opposed to the unfavorable association for the two other types is in line with findings from Denmark [5,7]. One explanation might be that wine drinkers drink less alcohol in total. In this study, wine drinkers (wine only or in combination with beer or liquor) reported a daily average of 1.07 units/d. Correspondingly, spirit drinkers reported 1.25 units/d and beer drinkers reported 1.17 units/d, on average. Findings in a study of U.K. women suggested that drinking alcohol with meals had a lesser effect on liver cirrhosis than drinking at other times [24]. The Norwegian drinking pattern, however, is far from being continental. Only 1% reported to drink wine with the dinner, as compared with 36% in Italy [25]. There have been reports of nonalcoholic substances in wine, such as resveratrol, which is found particularly in red wine that may reduce steatosis and hepatic fibrosis [26–28]. There may also be a link to the degradation of aflatoxin B_1, a strong hepatic toxin. Inoue et al showed that the risk of aflatoxin B_1 contaminant carryover was present in beer fermentation but reduced in wine fermentation because of hydration [29].

There was an intriguing risk-ratio modification between coffee and alcohol consumption on the liver cirrhosis mortality. If this finding is generalizable, one would expect a weaker association between alcohol consumption and liver cirrhosis in populations with high consumption of coffee or a weaker association between coffee consumption and liver cirrhosis in populations with low alcohol consumption. Norway is a country with comparatively high coffee consumption and low alcohol consumption [30,31]. Norway also has the lowest mortality of liver cirrhosis among the Western European countries [2].

Some studies have reported a favorable effect of coffee consumption on liver enzymes [32–34]. Klatsky et al [8] studied the coffee-cirrhosis association at different levels of alcohol consumption in persons with the highest 5% levels of aspartate aminotransferase and alanine aminotransferase, respectively. In both groups, there was an indication of a stronger coffee-cirrhosis association at higher levels of alcohol intake. These findings in persons with high transaminase enzymes levels fit in with our finding of a risk-ratio modification between alcohol and coffee consumption on ALD.

As a high coffee-consuming country, we could estimate risks at higher consumption levels than is usually done. The meta-analyses included studies with greater than or equal to 2 or greater than or equal to 3 cups/d as the highest categories [3,12]. Our data are able to extend on these results and show that there is a leveling off in risk. Above 5 or so cups/d, there was no further decrease in risk.

Our results strongly suggest that type of alcohol play a role. Coffee and alcohol drinking pattern vary across populations; however, and we are therefore reluctant to generalize our results, but the potential importance implies that they warrant further research.

Strengths and limitations

A strength of the study is a large study population with complete follow-up with respect to emigration and mortality. Cause of death was from the official certificates completed by doctors who are required to complete such certificates. These certificates are prepared in accordance with the International Classification of Diseases and Related Health Problems, World Health Organization. The cause of death was not further validated. In this study, 47 patients died of ALD in hospital, 35 patients died at home, and 5 persons died in nursing homes.

Table 5
Hazard ratios with 95% confidence intervals for mortality from alcoholic liver disease

Variable	HR* (95% CI)
Coffee (per category†)	0.63 (0.50–0.81)
Beer (per category‡)	2.06 (1.62–2.61)
Wine (per category‡)	0.68 (0.46–1.00)
Liquor (per category‡)	2.54 (1.92–3.36)

Men and women aged 30–67 years.
* Adjusted for sex, age, total cholesterol, triglycerides, smoking, body mass index, systolic blood pressure, education, and ill health.
† Coded as 0 = 0, 1 = 1–4, 2 = 5–8, 3 = ≥ 9 cups/d.
‡ Coded as 0 = 0, 1 = >0–<1.0, 2 = 1.0–<2.0, 3 = ≥ 2 units/d.

One of the limitations of the study is that we have only mortality data and not incidence data. Another limitation is that there has been no validation of the alcohol questions. The intake of alcohol during the last 14 days may be influenced by recall bias and it may also vary across the year, particularly during holidays and Christmas time. Neither are the coffee questions validated, but it is reasonable to assume that questions about daily intake (coffee) are less subject to misclassification than questions about intake over a longer period (alcohol). This was the finding in a Norwegian validation study in women, where the Spearman correlation coefficient between questionnaire and 24-hour recall was 0.82 for coffee and 0.67 for alcohol [35]. Finally, we have alcohol measurement at only one occasion. People may have changed their drinking habits during follow-up. Since the early 1990s, alcohol consumption has increased by approximately 40% in Norway [36]. The consumption of alcohol has also been changing to more wine and less liquor [25]. People who quit drinking completely are likely do so due to some health conditions. We do not know the impact on the alcohol-liver cirrhosis association, if any, of the possible changes in alcohol consumption during follow-up. Furthermore, we do not know whether the participants have changed their drinking habits before the screening due to digestive symptoms or a diagnosis of ALD. The exclusion of the first 3 years of follow-up is an attempt to lessen, but we cannot exclude the possibility of reverse causation. Finally, this is an observational study and we acknowledge the possibility of residual confounding.

Conclusion

Our finding supports a hypothesis of an inverse association between coffee and wine consumption and ALD mortality. Total alcohol consumption was adversely associated with ALD death, and the strength of the association varied with the level of coffee consumption.

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