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DIFFERENT DRINKING PATTERNS FOR WOMEN AND MEN WITH ALCOHOL DEPENDENCE WITH AND WITHOUT ALCOHOLIC CIRRHOSIS

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Abstract — Aim: The aim of our study was to investigate if there were differences in drinking patterns in patients with alcohol dependence (AD), with or without cirrhosis. **Methods:** We examined three groups in regard to differences in drinking patterns. We collected information from 50 patients with alcoholic cirrhosis (AC), 50 patients with AD, and 40 patients with non-alcoholic cirrhosis (NAC). We used the structured interview Lifetime Drinking History (LDH) to measure the alcohol consumed. Information regarding the total lifetime alcohol intake (LAI), drinking days (DD), drinks per drinking day (DDD), their beverage preferences, and their binge consumption was collected during interviews. **Results:** Women drank less than men. Women with AC reported 9,198 drinks as binge drinking compared to 25,890 drinks for women with AD without liver cirrhosis ($P < 0.05$), Women with AC reported 14,009 drinks of alcohol consumed during their lifetime compared to 45,658 drinks consumed by men with AC ($P < 0.0001$). Women with AD had drunk 5.8 DDD, and men had 8.5 DDD ($P < 0.05$). Both women and men with AC had significantly fewer DDD compared to men and women with AD without cirrhosis, 4.4 drinks for women ($P = 0.046$) and 6.2 for men ($P = 0.048$) with AC. **Conclusions:** Patients with AC seem to be predisposed to the hepatotoxic effects of alcohol- and the affected women seem to be even more sensitized. Binge drinking, rather than continuous drinking, does not seem to be especially associated with the development of cirrhosis. That women had drunk less alcohol during binge drinking further emphasizes this.

INTRODUCTION

The risk of acquiring alcoholic cirrhosis (AC) has been suggest to increase when the alcohol intake increases (Substance Abuse and Mental Health Administration, 2005). In a case-control study, the risk of acquiring cirrhosis increased when the alcohol consumption exceeded 40 g per day (Batey *et al*., 1992). A cohort study found an increased risk of cirrhosis with increasing alcohol intake (Klatsky *et al*., 1993). From cross-sectional, case-control, and prospective cohort studies a threshold has been observed (Corra *et al*., 1998). Only a few individuals who have a high alcohol intake will develop cirrhosis. A population study found a frequency of 4.2% of alcohol liver disease among individuals consuming 60 g or more of alcohol per day (Bellentani *et al*., 1997).

In epidemiological studies, an association has been observed between the consumption of the different alcoholic beverage and the risk of developing cirrhosis. Spirits consumption has been associated with liver cirrhosis mortality (Roizen *et al*., 1999; Stokkeland *et al*., 2006). There have been reports of a decreased risk of liver cirrhosis when a large part of alcohol was consumed as wine (Becker *et al*., 2002), but the reduced risk of developing liver cirrhosis with predominant wine consumption were not confirmed in a case-control study (Pelletier *et al*., 2002).

No earlier study has evaluated the cumulative drinking pattern and volumes in patients with end-stage AC and compared them to the pattern in patients with longstanding alcohol dependence (AD) to evaluate whether there are differences in lifetime alcohol intake (LAI) that may play a role in the development of alcoholic liver disease. To evaluate alcohol consumption during the whole lifetime the structured interview Lifetime Drinking History (LDH) is widely used (Skinner, 1982).

The aim of this study was to elucidate whether alcohol dependent patients with AC had a specific drinking pattern or a volume of consumption of alcohol, which differed from patients with AD without liver disease.

MATERIALS AND METHODS

Subjects

Patients currently in treatment for AD, alcohol-related, and non-alcohol-related liver cirrhosis were invited to participate. Fifty patients with AC and 40 patients with non-alcoholic cirrhosis (NAC) were recruited from the Department of Gastroenterology and Hepatology, Karolinska University Hospital in Stockholm and from the Department of Internal Medicine, Visby Hospital in Visby between October 1999 and November 2003. These patients were consecutively recruited from the outpatient service and from among patients hospitalized with liver cirrhosis at the departments (Table 1). Fifty patients with AD were recruited from the Magnus Huss Clinic, Karolinska University Hospital, and the Alcohol Clinic, Visby Hospital, during the same period. These patients were consecutively recruited from an outpatient relapse prevention program at the Magnus Huss Clinic, Karolinska Hospital and the Alcohol Clinic, Visby Hospital, and they had all been diagnosed having AD according to the DSM-IV criteria (Diagnostic and Statistical Manual of Mental Disorders, 1994). They denied all forms of chronic liver disease, and showed no clinical or biochemical signs of chronic liver disease. They all denied illicit drug use.

There were 13 women and 37 men with AC, 9 women and 41 men with AD, and 23 woman and 17 men with NAC. Men

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Table 1. Laboratory findings in patients with alcoholic cirrhosis (AC), alcohol dependence (AD), and Non-Alcohol Cirrhosis (NAC) included in the study. The Child-Pugh grade is presented for patients with liver disease

Women

	AC ($N = 13$)	AD ($N = 9$)	NAC ($N = 23$)
Age (min–max)	60.2 (54–73)	54.7 (39–75)	60.9 (23–83)
BMI (kg/m^2)	21.9 (±1.8)	21.1 (±0.8)	24.7 (±2.3)
Child-Pugh grade A	2	n.a.	12
Child-Pugh grade B	8	n.a.	8
Child-Pugh grade C	6	n.a.	3
Hemoglobin (g/l)	122 (±10)	130 (±9)	124 (±8)
WBC (x 10^9/l)	6.7 (±5.2)	8.5 (±0.7)	5.8 (±0.9)
Platelets (x 10^9/l)	169 (±38)	284 (±63)	159 (±46)
INR	0.9 (±0.1)	1.3 (±0.3)	1.2 (±0.2)
Albumin (g/l)	38.0 (±3.7)	34 (±5.3)	36.2 (±2.6)
Bilirubin (umol/l)	25.0 (±20.4)	14	26.6 (±20.2)
ASAT (ukat/l)	0.9 (±0.4)	0.8 (±0.4)	1.6 (±0.8)
ALAT (ukat/l)	0.5 (±0.2)	0.6 (±0.3)	0.8 (±0.3)
ALP (ukat/l)	5.3 (±1.4)	2.9 (±0.4)	7.2 (±2.0)
γ-GT (ukat/l)	3.5 (±2.9)	0.75 (±0.3)	4.2 (±2.0)

Men

Groups	AC ($N = 37$)	AD ($N = 41$)	NAC ($N = 17$)
Age (min–max)	58.7 (36–73)	54.5 (38–69)	60.1 (36–78)
BMI (kg/m^2)	21, 9 (±2.1)	24.0 (±1.5)	26.8 (±1.7)
Child-Pugh grade A	6	n.a.	8
Child-Pugh grade B	15	n.a.	7
Child-Pugh grade C	16	n.a.	2
Hemoglobin (g/l)	126 (±10.1)	148 (±5.4)	140.6 (±7.1)
WBC (x 10^9/l)	6.6 (±1.8)	7.4 (±0.8)	5.8 (±0.5)
Platelets (x 10^9/l)	143 (51)	266 (±41)	170 (±34)
INR	1.3 (±0.3)	1.09 (±0.06)	1.1 (±0.01)
Albumin (g/l)	32 (±4.3)	39.3 (±2.4)	36.3 (±2.9)
Bilirubin (umol/l)	29.0 (±40.4)	16.8 (±3.4)	23.4 (±7.1)
ASAT (ukat/l)	1.0 (±0.7)	0.9 (±0.4)	1.0 (±0.1)
ALAT (ukat/l)	0.7 (±0.4)	0.6 (±0.2)	0.7 (±0.1)
ALP (ukat/l)	4.2 (±1.5)	3.36 (±0.5)	6.9 (±2.8)
γ-GT (ukat/l)	4.5 (±3.1)	2.5 (±2.09)	5.1 (±3.8)

with AC had a mean age of 58.7 years, and the mean age for men with AD was 54.5 years. Men with NAC had a mean age of 60.1 years. Women with AC had a mean age of 60.2, and the women with AD had a mean age of 54.7. The mean age of women with NAC was 60.9 years. Three patients with AC and two patients with NAC refused to participate. Ten of the patients with AC had Child-Pugh's grade A, 23 had grade B, and 17 had grade C (Pugh *et al*., 1973). Twenty-two patients with NAC had Child-Pugh grade A, 14 had grade B, and 4 had grade C. None of the patients had encephalopathy at the time of interview although two patients with NAC and seven patients with AC had medication indicating previous episodes of encephalopathy (per oral lactulose).

The diagnosis of cirrhosis was based on clinical signs of complications due to liver disease, such as ascites, hepatic encephalopathy, or oesophageal varices, or by persistent biochemical abnormalities indicating cirrhosis: 6 months or more with spontaneously prolonged international normalized ratio (INR) or a lowered serum albumin level. Liver biopsy had been performed on four of the patients with AC, and on 26 of the patients with NAC. All biopsies confirmed the diagnosis of cirrhosis. Exclusion criteria were hepatitis B or C, terminal disease, psychiatric disorders which made interviewing impossible, or untreated encephalopathy.

All patients with AC had been hospitalized previously for AD. The diagnosis of AC was based on clinical findings of decompensated liver cirrhosis. Ascites was found in 33 patients, 15 patients had oesophageal varices that had bled, and 6 patients had oesophageal varices without bleeding. Among the patients with oesophageal varices without bleeding, four patients had severe ascites, and two patients had a previous history of encephalopathy. All patients with AC had symptoms and signs that were related to their cirrhosis (i.e. either previous or current oesophageal varices, encephalopathy or ascites).

The patients with NAC had been diagnosed as having the following disorders: 13 patients with hemochromatosis, 11 patients with primary biliary cirrhosis, 7 with autoimmune hepatitis, 6 with cryptogenous cirrhosis, 2 with primary sclerosing cholangitis, and 1 patient with Wilson's disease. Ascites were found in 11 patients, 6 patients had oesophageal varices that had bled, and 10 patients had oesophageal varices without bleeding.

The patients with AD had all been evaluated by a psychiatrist and were diagnosed with AD according to both ICD-10 and DSM-IV. They denied having a history of liver disease and had no biochemical markers of viral or chronic liver disease, and had never had any signs or symptoms of liver disease.

All patients had their blood count, liver function tests, and liver enzymes registered at the time of their interview.

The study was approved by the Karolinska Institutet Ethics Committee, and was conducted in accordance with the Declaration of Helsinki.

Lifetime drinking history assessment

The LDH was used to provide quantitative indices of past alcohol consumption. We translated the Dutch form into Swedish, and designed it to be scanned and interpreted by computer software (Lemmens, personal communication 1997). The interviews were performed at the Magnus Huss Clinic in Stockholm and at Visby Hospital. All interviews were conducted and the forms filled out by one of the authors (KS). Information about the patient's liver disease was taken from patient files and confirmed during the interview, and biometrics and sex were registered. Binge drinking was defined as five or more drinks per day.

Statistics

Overall differences in drinking parameters between the three patient groups were analysed using the Kruskal–Wallis test

combined with exact Wilcoxon posthoc tests to detect differences between groups (Kruskal and Wallis, 1952; Siegel and Castellan, 1998). The *P*-values from the paired comparisons were adjusted by the Holm method (Holm, 1979). All values were expressed as the mean and first and third quartile. Results are presented separately for women and men (Table 2).

RESULTS

The onset of alcohol intake (OA), duration of alcohol consumption (DAC), drinking days (DD), and the drinks consumed per drinking day, the total lifetime alcohol intake (LAI), beverage types consumed, and the volume of alcohol drunk as binge drinking for all patient groups are shown in Table 2.

Patients with NAC did not differ significantly from the patients with AC and patients with AD with regard to the onset age of alcohol consumption and DAC. However, they had significantly less DD, drinks per drinking day (DDD), total lifetime intake of beer, wine, and spirit. The volume of alcohol drunk as binges was significantly lower compared to the two other patient groups ($P < 0.01$ for all comparisons, both for men and for women).

The mean DAC was 36.6 years for women with AC and 34.6 year for women with AD without cirrhosis. Men with AC had been consuming alcohol for 39.0 years, and men with AD without cirrhosis had been consuming alcohol for 37.0 years. There were no significant differences between women and men with AC, and women and men with AD without cirrhosis ($P = 0.43$ and $P = 0.23$). There were no significant differences for women and men with alcohol cirrhosis compared to women and men with AD without cirrhosis ($P = 0.65$ and $P = 0.139$). The number of days and number of years the two patient groups were exposed to alcohol were the same.

Women with AC drank 4.4 DDD compared to 5.8 drinks for women with AD without cirrhosis ($P < 0.01$). Men with AC had consumed 6.2 DDD as compared to men with AD without

Table 2. Measures of drinking pattern for patients with alcoholic cirrhosis (AC), alcohol dependence (AD), and non-alcoholic cirrhosis (NAC) are presented: the age of the first drink (onset age), and the duration of alcohol consumption are presented as years. The number of drinking days, the drinks per drinking day, the total lifetime alcohol intake are expressed as number of standard drinks, and the lifetime intake separated into beer, wine and spirits, and the amount drunk during binges are presented as number of standard drinks. Overall differences are presented using the Kruskal–Wallis test combined with exact Wilcoxon posthoc tests for differences between groups

Women

	AC (Q1–Q3)	AD (Q1–Q3)	*P*-value (AC vs AD)	NAC (Q1–Q3)	*P*-value (total)
Onset age	20.9 (18.0–20.0)	19.6 (17.0–19.5)	–	20.8 (16.3–21.8)	0.43
Duration of alcohol consumption	36.6 (35.0–41.0)	34.6 (30.5–35.9)	–	29.7 (15.0–47.0)	0.43
Drinking days	4344 (2,215–5,222)	4,900 (2,714–6,327)	0.65	2,141 (70–2,408)	0.002**
Drinks per drinking day	4.4 (2.0–4.0)	5.8 (3.8–6.8)	0.0046**	1.7 (1.0–2.0)	<0.0001**
Lifetime alcohol intake	14,010 (6,735–14,040)	27,440 (21,180–41,470)	0.09	4,574(111–4,348)	<0.0001**
Beer	1,611 (0–2,096)	11,350 (7,529–16,350)	0.002**	1,206 (0–635)	0.0002**
Wine	6,845 (4,165–9,360)	10,020 (6,707–14,990)	0.23	2,561 (44–2,918)	0.0002**
Spirits	5,554 (2,000–5,145)	6,075 (2,615–9,296)	0.31	807 (10–1,247)	<0.80**
Binge	9,198 (0–10,940)	25,890 (18,840–39,360)	0.049**	921 (0–0)	<0.0001**

Men

	AC (Q1–Q3)	AD (Q1–Q3)	*P*-value (AC vs AD)	NAC (Q1–Q3)	*P*-value (total)
Onset age	18.2 (15–19)	16.5 (15–19)	N. S.	17.4 (15–19)	0.07
Duration of alcohol consumption	39.0 (34.0–44.0)	37.0 (32.0–43.3)	N. S.	41.4 (36–48)	0.29
Drinking days	7,327 (4,952–10,260)	6,272 (3,159–7,738)	0.139	2,729 (819–3,790)	<0.0001*
Drinks per drinking day	6.2 (4.0–9.0)	8.5 (4.75–10.25)	0.048**	4.0 (2.0–6.0)	<0.001*
Lifetime alcohol intake	45,660 (22,200–57,280)	49,760 (23,310–50,760)	0.78	11,120 (3,582–17,330)	<0.0001*
Beer	17,580 (7,763–19,680)	20,690 (5830–17,350)	0.33	4,143 (359–5,102)	0.0001*
Wine	12,380 (3,360–14,500)	13,250 (4,207–15,410)	0.31	2,716 (2,630–13,670)	0.0007*
Spirits	15,700 (5,400–16,080)	15,820 (5,205–16,290)	0.63	4,263 (1,027–4,135)	<0.0002*
Binge	41,150 (10,320–58,000)	47,890 (21,090–49,490)	0.35	9,104 (135–43,740)	<0.0001*

The *P*-values from paired comparisons were adjusted by the Holm method. All values were expressed as the mean ± SD $* = P < 0.05$, $** = P < 0.01$.

cirrhosis who had been drinking 8.5 DDD ($P < 0.05$). All patients with AC drank significantly less DDD compared to patients with AD.

Women with AC had drunk 14,010 drinks during their lifetime, whereas women with AD without cirrhosis had drunk 27,440 drinks, although the difference between the mean values was large it did not reach statistical significance ($P = 0.09$). Men with AC had drunk 45,660 drinks during their lifetime, and men with AD without cirrhosis had drunk 49,760 drinks, which did not differ significantly ($P = 0.78$). The LAI did not differ significantly between all patients with AC and patients with AD without cirrhosis.

A large difference was seen when comparing women with AC to women with AD without cirrhosis when we analysed the total amount of alcohol they had consumed as beer during their lifetime. Women with AD without cirrhosis had drunk 11,350 glasses of beer compared to women with AC who had drunk 1,611 glasses ($P < 0.01$). There was no difference in the beer consumption between men with alcohol cirrhosis compared to men with AD without cirrhosis ($P = 0.33$). There were no differences between patients with AC and the patients with AD without cirrhosis concerning the consumption of wine and spirits (for men: $P = 0.31$ for wine and $P = 0.63$ for spirits, and for women: $P = 0.23$ for wine and $P = 0.31$ for spirits). Women with AC had drunk significantly fewer drinks of beer than women with AD without cirrhosis.

The measures of alcohol consumption by men with alcohol cirrhosis found in Table 2 are used to compare the consumption of alcohol by women with alcohol cirrhosis, which is presented in Table 2. These comparisons are presented in Fig. 1. Women had a later onset of alcohol consumption compared to men ($P = 0.04$). Women consumed less alcohol during their lifetime than men ($P < 0.0001$). The total consumption of beer was significantly less for women compared to men with AC ($P < 0.0001$), as well as for spirits ($P = 0.0009$), but there was no significant difference regarding the consumption of wine ($P = 0.776$). Women with alcohol cirrhosis drank less alcohol during binge drinking than men ($P < 0.0004$). In addition–which is not seen in the figure–women had fewer DD ($P = 0.007$), and drank fewer DDD ($P = 0.014$), and their drinking onset ages were significantly later than men ($P = 0.04$), although both women and men had a similar DAC ($P = 0.99$).

DISCUSSION

The aim of this study was to examine patients with the end-stage of alcohol-related liver disease, i.e. assessed cirrhosis, and to compare the drinking pattern in these patients to the drinking pattern in patients with AD without cirrhosis, retrospectively. This implies that we could not select the patients from the same patient population. The selection was not equal, and some differences found between the three groups might be a matter of different selection of the patients. People with AC may avoid health care, and the ones with the gravest symptoms and signs may not survive to be interviewed. Alternative study designs could be used to explore whether the differences in drinking patterns could influence the development of AC. A prospective approach could add additional value, albeit expensive and time-consuming, as the development of cirrhosis takes decades (Bellentani and Tiribelli, 2001). However, all patients received information regarding the study in which both patients with AC and the alcohol dependent

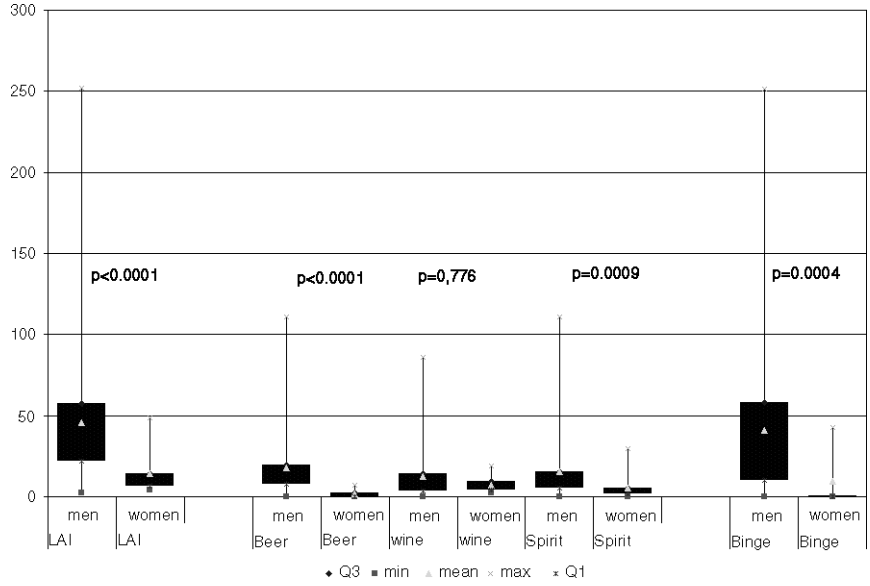

Fig. 1. The drinking pattern for men compared to women with alcoholic cirrhosis: the total lifetime alcohol intake (expressed as number of standard drinks), the lifetime intake consumed as beer, wine, and spirits, respectively, and the amount drunk during binges. Values are presented as mean, first quartile (Q1) and the third quartile (Q3), and the minimum and maximum value. There were significant differences between men and women regarding their lifetime alcohol intake (LAI), $P < 0.0001$, the lifetime intake of beer, $P < 0.0001$, the lifetime intake of spirits, $P = 0.0009$ and the volume alcohol consumed as binge, $P = 0.0004$. There were no significant difference regarding the consumption of wine, $P = 0.776$. The lifetime indexes are presented as thousand drinks in the figure.

patients' problems were related to excessive alcohol consumption. All patients with AC and AD without cirrhosis accepted this description of their problems prior to the interview. We think that the selection of the patients was as uniform as possible so as to elucidate differences in lifetime drinking pattern in the groups studied.

The LDH questionnaire has been reliability-tested, and generally, has better reliability when administered as an interview rather than a self-administered questionnaire (Lemmens *et al*., 1998; Friesma *et al*., 2004). The reliability of the LDH interview is reported to be good from 0.78, when compared to interview data from spouses. When tested after approximately a 5-month interval using the LDH questionnaire interview with time intervals that the patients defined, the correlations for the duration of drinking were 0.94, 0.80 for drinking volume and 0.68 for the daily average (Lemmens *et al*., 1998). Even after 5 years the reliability is reported to be good (Jacobs *et al*., 2006). Biased answers may occur even if the questionnaire used is reliable. All patients with AC had been hospitalized at least once for AD, thus, we think that the recall bias may play little role for the differences found in the drinking patterns between patients with AD and AC, and patients with AD without cirrhosis. To enhance reliability we developed an interview-based LDH. We think that the interview and technique used when interviewing the patients were as thorough as possible to render the most valid report of prior consumption of alcohol.

The main finding in this study was that men and women with AC did not consume more alcohol than the patients with AD without cirrhosis. For women there was even a less LAI, but the difference did not reach statistical significance. This may support the view that there exists a threshold effect for alcohol to elicit liver damage (Kamper-Jorgensen *et al*., 2004; Corrao *et al*., 2004). It could also be an effect of the selection of the patients. We interviewed consecutive patients who had survived after being treated for symptoms of liver decompensation as ascites and bleeding from oesophageal varices. A lower reported alcohol consumption in our patients with AC could be influenced by the tendency of patients with decompensated cirrhosis to report less alcohol consumed to be accepted for interventions regarding their liver disease. We think our patients with AC are representative for patients with symptoms of cirrhosis with decompensation in Sweden as the age and sex distribution follow the epidemiological data we have on hospitalized patients with liver disease (Stokkeland *et al*., 2006). All our patients with AC had been hospitalized diagnosed with AD. This implies either that all patients had AD or that the diagnosis of dependence was made on the basis of their liver disease. A careful investigation into degree of AD in patients with AC found that the majority did not score more than 'mild' on dependence (Smith *et al*., 2006). In addition, at least 22 of the patients with AC died after the interview, implying that we did not select a group of patients with a more benign prognosis than generally found among Swedish patients with decompensated AC. The male patients with AC had drunk similar amounts of alcohol per year compared to alcohol consumed by the Swedish patients with hepatitis C between two biopsies (Westin *et al*., 2002). We think that our findings support the hypothesis that alcohol consumption is not increased in patients with AC compared to patients with AD without cirrhosis.

As expected, the patients with NAC had been drinking less alcohol than both patients with AD with and without cirrhosis.

All patients with AC drank fewer DDD than patients with AD without cirrhosis. Women with AC drank less than men with AC except for wine consumption, and they drank less than women with AD without cirrhosis, although the difference in LAI did not reach statistical significance. This highlights the possible existence of an increased susceptibility to alcohol-induced liver damage in women compared to men, and this is well in line with earlier cohort studies (Corrao *et al*., 1998). The increased susceptibility to alcohol could be explained by the interaction of alcohol with the female sex hormone. It is known that estrogen enhances the immune response in women, and this enhancement could increase the female risk for developing cirrhosis (Kovacs and Messingham, 2002). We acknowledge the fact that there were only 13 women with AC, and that this might impair the validity of our conclusions regarding the differences we found in drinking patterns. That there were few women compared to men with AC included in our study is in line with the relation we found between men and women hospitalized for liver diseases in Sweden (Stokkeland *et al*., 2006).

Women with AC drank less as binge drinking, and this is opposed to a report from the United Kingdom which highlighted a temporal association between the increase in heavy sessional drinking and the increase in mortality in liver cirrhosis (Pincock, 2003). The observed difference in binge drinking adds strength to the suggested hypothesis, that women may be more susceptible to the hepatotoxic effects of alcohol than men. In men, there was no difference in the proportion of drinking as binge drinking between the alcohol dependent men with AC and those without cirrhosis. Our data therefore does not indicate that binge drinking is more hepatotoxic than consumption of a lower volume of drinks per drinking occasion, and opposes the conclusions based on effects of binge drinking observed under experimental conditions in cirrhotics (Abril *et al*., 1999; Oekonomaki *et al*., 2004).

In conclusion, we found that patients with AD and AC did not drink more alcohol than patients with AD without cirrhosis. Thus, patients who develop AC are presumably predisposed to be sensitive to the hepatotoxic effects of alcohol. Women with AC had been drinking less both when compared to women with AD without cirrhosis and compared to men with AC, further supporting the hypothesis of sensitization towards alcohol, and emphasizing the additive effect of the female gender to the negative effects of alcohol in the predisposed individuals. Further studies with different methodological approaches are of interest to elucidate the nature of an increased susceptibility to the hepatotoxic effect of alcohol, especially in women.

LIFETIME DRINKING HISTORY ASSESSMENT

The LDH was used to provide quantitative indices of past alcohol consumption. We translated the Dutch form into Swedish, and designed it to be scanned and interpreted by computer software (Lemmens, personal communication 1997). The interviews were performed at the Magnus Huss

Clinic in Stockholm, and at Visby Hospital. All interviews were conducted, and the forms filled out by one of the authors (KS). Information about the patient's liver disease was taken from patient files and confirmed during the interview, and biometrics and sex were registered.

Information was given as to which types of beverage the interview included, and the definition of a standard drink of alcohol for each beverage type. One standard drink being the equivalent of 12 g of pure alcohol which is the equivalent to 50 cl of beer containing 3,5% alcohol, 33 cl beer containing 4,5% alcohol, 14 cl wine, 8 cl fortified wine or 4 cl distilled liquors. The OA was registered, and identical questions were asked for the age intervals from 12 to 18, 19–27, 28–44, 45–60, and for age above 60 years. The patients were asked about quantity, frequency, beverage types, binge drinking, and periods of abstinence. Binge drinking was defined as drinking five or more drinks in one day both for men and women. Questions were asked concerning the alcohol consumption during the year and week prior to the interview. The interview took about 30 min.

After all interviews were completed, each form was scanned to make the responses available for computer analysis.

Based on the answers in the questionnaire the following parameters were available for analysis: age, gender, body mass index (BMI) and OA. The DAC was defined as the time period from the onset age to the end of drinking. DD were all days the patients consumed alcohol throughout all age groups. The LAI was the sum of all alcohol consumed during the patient's life. The average number of DDD was defined as the mean number of DDD for patients throughout all age groups.

For the purpose of calculation, the frequency of alcohol consumption was expressed as days per year. The answer 'every day per week' was defined as 360 days, '5–6 days per week' as 280 days, '3–4 days per week' as 180 days, 'once or twice per week' 80 days, '1–3 times per month' 24 days, '3–5 days per half-year' 7 days, '1–2 days per half-year' 3 days and 'more seldom than 1–2 days per year' 0 days.

To quantify the consumption of different beverage types the patients were asked to separately estimate the consumption of beer, wine, and spirits during each age interval as 'always', 'often', 'sometimes', 'seldom', and 'never'. The answers were given the values 1.0 for 'always', 0.75 for 'often', 0.5 for 'sometimes', 0.25 for 'seldom', and 0 for 'never'. The fractions of alcohol consumed as beer, wine, and spirits were then calculated for all age intervals.

Binge drinking was expressed as the total volume of alcohol consumed during binges using the binge frequency answers multiplied by five drinks. If the patient had answered that the average number of drinks during the period was more than five the volume of binge was calculated by multiplying with that amount of drinks.

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