Smoking decreases stimulation by alcohol, parental history of alcoholism does not

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Abstract

Objective. Low stimulation by alcohol is thought to be a risk factor for alcoholism because it is more common among sons of alcoholics than among other men. The aim was to find out which is more important determinant of low stimulation by alcohol, parental history of alcohol problems or exposure to tobacco.

Subjects and methods. Cross-sectional self-report data on 512 subjects from a representative community sample and on 167 consecutive hospital patients with alcohol dependence were pooled.

Results. Stimulation by alcohol correlated negatively with the number of cigarettes smoked daily (r = -0.25; p<0.001). In multivariate regression analysis, self-reported stimulation by alcohol was negatively related to the number of cigarettes smoked daily, age and alcohol intake (p<0.001 for all), and was higher among men than among women (p=0.03). Parental history of alcohol problems was not significant (p=0.6). The analysis suggests that the average person smoking 40 cigarettes a day will experience 20% less stimulation from drinking alcohol compared with a no-smoker.

Conclusions. Smoking may explain why sons of alcoholic fathers have been found to have lower response to alcohol and higher risk for alcoholism than other men.

Introduction

Familial history of alcoholism increases the risk of the offspring (1). Sons of alcoholic fathers have been found to have lower subjective response after alcohol ingestion than sons of other fathers (2, 3). In most studies, the measure of responsivity has been subjective feeling of stimulation by alcohol, such as being Ahighε or Aintoxicatedε (2). These findings have been interpreted as evidence for an innate genetic tolerance to alcohol among high-risk subjects. However, human experiments showing that acute administration of nicotine decreases the subjective effects of alcohol (4) suggest an alternative hypothesis: tobacco use decreases the subjective response to alcohol. This, in turn, might increase alcohol intake and subsequent risk of alcohol dependence among those who aim at experiencing the intoxicating effects of alcohol. Some support to this hypothesis is provided by an Australian twin study (5). It found that, compared with non-smokers, self-reported current smokers had lower scores of subjective intoxication after alcohol challenge even after controlling for self-reported alcohol intake (5). However, the Australian study did not inquire into parental history of alcoholism. The US study finding lower subjective response after alcohol ingestion among sons of alcoholic fathers than among other sons did not permit to analyse the role of tobacco because the study groups were matched on smoking (3, 6). At present, it is not clear which is more important determinant of low stimulation by alcohol, exposure to tobacco smoke or parental history of alcohol problems. The present study aims to clarify this question.

Subjects and methods

Subjects
To ensure adequately large variance in variables under study, two cross-sectional data
sets were pooled, 548 subjects free from alcohol dependence from a representative community sample and 305 subjects with the diagnosis of alcohol dependence from a clinical series of consecutive patients in a hospital specializing in the treatment of addiction. Sampling and characteristics of the subjects have been described earlier (7). For the present analysis, all 174 persons with missing values in any of the variables under study were excluded. This left 679 subjects for analysis. There were 512 subjects (234 men) from the community sample and 167 subjects (73 men) from the clinical sample.

**Measures**

Anticipated Biphasic Alcohol Effects Scale (BAES) was used to assess self-reported intensity of alcohol-induced stimulation and sedation during the last recalled instance of alcohol intoxication. Both scales contained 7 items on 11-point Likert ratings (from "not at all" to "very much"). BAES has been found to have high internal consistency during both the ascending and descending limbs of the blood alcohol curve (8). Its factor structure had been confirmed and scores for stimulation have been found high during the ascending limb of the blood alcohol curve (9).

Tobacco smoking was assessed by inquiring into the usual number of cigarettes (including self-rolled ones), cigars and pipes per day. No adjustments to correct for the variation in nicotine content were possible.

Alcohol intake was assessed by inquiring into the usual frequency and amount of consuming beer, wine and spirits during the past 12 months. In populations where alcohol intake varies much over time this approach gives more reliable estimates on long-term intake than relying on shorter recall periods (10). Estimates of mean alcohol intake were based on the following average alcohol contents by volume: beer 5.0 percent, natural wine 11.6 percent, fortified wine 18.7 percent and spirits 37.0 percent.

Parental history of alcohol problems was assessed by asking whether the parents of the respondents had ever had alcohol problems or had been in treatment because of these. The test-retest reliability for classification of first degree relatives has been found to be good, while data for second-degree relatives is considerably less reliable (11).

Questions used to operationalize the diagnosis of alcohol dependence according to both ICD-10 criteria have been described earlier in detail (12). There were altogether 10 questions. These questions pertained to the period of past 12 months.

**Statistical analysis**

Scatterplots and histograms were used to visually examine the distribution of the variables under study. The significance of the differences between means was assessed by the t-test or the F-test and that of associations between categorical variables by the Chi-square test. Pearson bivariate correlation coefficient was applied to examine correlations between two continuous variables and multivariate regression analysis to examine relation of the independent variables to the dependent one.
Results

The 174 subjects with missing values in any of the variables under study were excluded. Compared with the former, persons included in the analysis were more often men (45.2% vs. 33.3%; Chi-square test p=0.005) and had less often alcohol dependence (24.6% vs. 79.3%; Chi-square test p<0.001). No differences were found for mean age (42.9 vs. 41.9 years; t-test p=0.3) or proportion of those married (37.4% vs. 44.8%; Chi-square test p=0.07). The characteristics of the included subjects by alcohol dependence are shown in Table 1. Heavy smoking was rare in the community sample but common among the alcohol dependence patients. Proportion of subjects smoking more than 20 cigarettes a day was 2.9% among the former, 25.1% among the latter group (Chi-square test p<0.001).

Table 1 approximately here

Self-reported stimulation by alcohol was approximately normally distributed among subjects. It correlated negatively with the number of cigarettes smoked daily (r=-0.25; p<0.001). Mean values decreased by the number of cigarettes smoked daily (Table 2). There was a significant positive correlation between alcohol intake and the number of cigarettes smoked daily (r=0.42; p<0.001).

Table 2 approximately here

In multivariate regression analysis (Table 3), self-reported stimulation by alcohol was negatively related to the number of cigarettes smoked daily, age and alcohol intake (p<0.001 for all), and higher among men than among women (p=0.03). Parental history of alcohol problems was not a significant predictor (p=0.6). There was no significant interaction between alcohol intake and the number of cigarettes smoked daily (p=0.6). No part of parental history was significantly related to stimulation by alcohol when its components were analysed separately (treatment for alcoholism, other alcohol problems, father, mother). The negative effect was of somewhat stronger among men (regression coefficient -0.312; SE 0.07; p<0.001) than among women (regression coefficient -0.218; SE 0.07; p=0.003), when analysed separately. However, no significant interaction was found between sex and the number of cigarettes smoked daily (p=0.2).

Table 3 approximately here

Discussion

Self-reported stimulation by alcohol was not related to parental history of alcohol. However, it associated with smoking in monotone dose-response fashion. The more
cigarettes smoked daily, the lower self-reported stimulation by alcohol. Thus, smoking seems to attenuate the subjective effects of alcohol. This relation was still observed after adjustment for age, sex and alcohol intake. Adjustment for alcohol is likely to control for the possible increase of both metabolic and central nervous system tolerance to alcohol and that for age for the possible age-related decrease.

The present study assessed self-reported stimulation by alcohol. Some of the earlier studies have assessed a mixed measure of low response to alcohol, consisting on both subjective feeling of intoxication and objective measurement of body sway (3, 5). The outcome variable thus differs between the studies. There seems to be no data on the effects of long-term smoking on body sway during alcohol challenge. Laboratory experiments have found that nicotine increases body sway among inexperienced smokers (13, 14). This suggests that the low responsivity to alcohol among smokers is not due the effects on body sway but to decreased subjective feeling of intoxication.

Selection bias should be considered. The community sample was representative, but if patients with high stimulation by alcohol would have been selected out from the hospital sample then selection bias could distort the actual association. This does not seem likely since high stimulation by alcohol tends to lead into more interpersonal and health problems and thus for more need for treatment than low stimulation. The internal validity of the study might be undermined by the fact that 20% of all subjects had to be excluded from analysis because of missing responses in one or more of the variables under study. However, these subjects did not differ from those included in the analysis by age or sex. Overrepresentation of women and subjects with alcohol dependence among the excluded are probably related to poor tenacity to answer questionnaires among some alcoholics and the preponderance of women in the hospital available for data collection (7).

The present study relied on self-reports, which are, of course, always less than fully reliable. Recall of alcohol intake, stimulation by alcohol and cigarettes smoked daily and other self-reported variables under study is likely to be inaccurate. However, errors in recall are likely to vary randomly. The data analysis did not reveal any inconsistencies that would have lead to suspect the existence of any systematic bias. The results also agree with laboratory experiments during acute administration of nicotine and alcohol (4). The present findings suggest that similar relations apply also during long-term consumption of these substances.

The magnitude of the subjective response after alcohol ingestion among sons of alcoholic fathers is not much lower than among sons of other fathers. The effect size has been considered to be small to moderate (2). The effect of smoking seems to be large. The model in Table 3 indicates that on the average, a person smoking two packs, i.e. 40 cigarettes, a day will experience 20% less stimulation from drinking alcohol compared with a no-smoker.

There is cross-sectional evidence showing that smoking and alcohol go together. In a nationally representative sample of almost 43,000 US adults, the higher the alcohol intake, the higher also the prevalence of smoking. Smoking was more common among
subjects with diagnosis of alcohol abuse or dependence than among those without such diagnosis (15). In the population-based St Louis Epidemiologic Catchment Area Survey the odds for DSM-III alcoholism (abuse and dependence) among ever-smokers was five-fold compared with no-smokers (16).

Follow-up studies have found that alcoholism and heavy use of alcohol is predicted by smoking and smoking by alcohol use already in adolescence (17). In a cohort of 940 adolescents, followed up for 6-10 years, daily smoking and conduct/oppositional defiant disorders predicted future alcoholism (abuse or dependence), when adolescent depression, alcoholism and other substance use disorders were controlled for (18). In a 5-year follow-up study with 86% of the 709 students reporting complete data, both high average alcohol intake and binge drinking (13 or more drinks on occasion) were significantly related to male sex and earlier high scores of relief drinking and relief smoking (19). Among a cohort of 14,130 non- to moderate drinkers, smoking predicted the onset of heavy drinking (20). Moreover, a follow-up study of young adults found that tobacco dependence is more common among persons with family history of alcoholism and that there seems to be a common - both genetic and environmental - vulnerability to tobacco dependence and alcohol use disorders (21, 22). Smokers seem to be in increased risk of hazardous drinking and alcoholism. Offspring of alcoholics may have genes that predispose to both alcohol intake and tobacco smoking (23).

In conclusion, self-reported stimulation by alcohol associated with smoking in a negative dose-response fashion. Low stimulation by alcohol may be due tobacco smoking. Smoking may explain why sons of alcoholic fathers have been found to have lower response to alcohol and higher risk for alcoholism than other men.

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References


<table>
<thead>
<tr>
<th></th>
<th>Community subjects without alcohol dependence</th>
<th>Hospital patients with alcohol dependence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Number of subjects</td>
<td>512</td>
<td>167</td>
</tr>
<tr>
<td>% of men</td>
<td>45.7</td>
<td>43.7</td>
</tr>
<tr>
<td>% of married</td>
<td>40.2</td>
<td>28.7</td>
</tr>
<tr>
<td>% with parental history of alcohol problems</td>
<td>24.2</td>
<td>49.1</td>
</tr>
<tr>
<td>Mean age, years</td>
<td>45.7</td>
<td>44.4</td>
</tr>
<tr>
<td>% of no-smokers</td>
<td>34.2</td>
<td>0</td>
</tr>
<tr>
<td>Mean number of cigarettes smoked daily</td>
<td>3.2</td>
<td>19.2</td>
</tr>
<tr>
<td>Mean alcohol intake g/day</td>
<td>37.9</td>
<td>140.0</td>
</tr>
<tr>
<td>Mean stimulation by alcohol score</td>
<td>45.7</td>
<td>33.2</td>
</tr>
</tbody>
</table>
Table 3 Regression of self-reported stimulation by alcohol on the number of cigarettes smoked daily and other significant covariates among 679 persons

<table>
<thead>
<tr>
<th>Independent variable</th>
<th>Regression coefficient</th>
<th>Standard error</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cigarettes, number/day</td>
<td>-0.267</td>
<td>0.052</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Alcohol intake g/day</td>
<td>-0.032</td>
<td>0.007</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Parental history of alcohol problems, yes</td>
<td>-0.574</td>
<td>1.069</td>
<td>0.6</td>
</tr>
<tr>
<td>Age, years</td>
<td>-0.176</td>
<td>0.036</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Sex, male</td>
<td>2.115</td>
<td>0.971</td>
<td>0.03</td>
</tr>
</tbody>
</table>
| Constant 53.354 (Standard error 1.869); $r^2 = 0.13$