

SHORT REPORT

Alcohol consumption and frontal lobe shrinkage: study of 1432 non-alcoholic subjects

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Abstract

Objectives—To evaluate the influences of chronic alcohol consumption on brain volume among social drinkers, as it is well known that alcohol misusers have a high risk of brain shrinkage.

Methods—Frontal lobe volumes on MRI were compared with the current alcohol habits of consecutive 1432 non-alcoholic subjects.

Results—After adjusting for other variables, age was found to be the most powerful promoting factor for the shrinkage with an odds ratio of 2.8 (95% confidence interval (95% CI) 1.23–3.06) for each 10 years of age. Regarding alcohol habit, 667 of the subjects were abstainers, and 157, 362, and 246 of the subjects were light (average 88.2 g ethanol/week), moderate (181.2 g/week), and heavy (418.1 g/week) drinkers, respectively. Moderate alcohol consumption did not increase the incidence of frontal lobe shrinkage (odds ratio 0.98; 95% CI 0.73–1.33), whereas heavy drinkers were at a higher risk compared with abstainers (1.80; 1.32–2.46). The contributory rate of alcohol consumption for frontal lobe shrinkage was 11.3%.

Conclusion—The brain tends to shrink physiologically with age. Heavy alcohol consumption seems to exaggerate this shrinkage in social drinkers. Moderate alcohol consumption does not seem to affect brain volume.

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Keywords: alcohol; social drinker; brain atrophy

Many studies on the effects of alcohol on brain morphometry have disclosed that a large amount of alcohol consumption can induce brain shrinkage.^{1–4} To evaluate the effects of alcohol on brain volume among social drinkers, we compared the findings of frontal lobe shrinkage on MRI with current alcoholic habits of consecutive 1432 volunteers.

Subjects and methods

The subjects of the study were employees of a large Japanese company and their family members (1061 men and 371 women). We assessed their medical histories and drinking and smoking habits with a questionnaire.

We divided the subjects into four categories based on their current alcohol habit: abstainers, light drinkers (drinkers who consumed alcoholic beverages less than three times a week), moderate drinkers (drinkers who consumed alcohol more than four times a week, but who consumed less than 14 units of Japanese alcohol consumption a week (1 unit=180 ml of Sake (ethanol content 14%, 14 units= about 350 g of ethanol a week), and heavy drinkers (drinkers who consumed 14 units or more a week). All participants underwent MRI with a 1.5 T MR unit (Signa Horizon, GE). Frontal lobe volume was evaluated with 3 mm thick axial T2 weighted images. Widening of the CSF space of the frontal surface was considered to indicate shrinkage of the frontal lobe. The extent of the shrinkage was classified into four categories based on the width of the CSF space: no shrinkage (≤ 2 mm), mild shrinkage (3–5 mm), moderate shrinkage (6–8 mm), and severe shrinkage (≥ 9 mm).

No subjects had medical histories of head injury, stroke, or other neurosurgical disorders. All statistical analyses were performed using the commercially available SPSS statistical package (SPSS Japan Inc). *p* Values <0.05 were regarded as significant.

Results

A total of 1432 subjects (1061 men and 371 women) was recruited for analysis. Of these, 236, 536, 433, and 277 were in their 30s, 40s, 50s, and 60s, respectively (mean 49.1 (SD 10.1) years old). Concerning their alcohol habits, 667 of the subjects (46.5%) were abstainers and 157 (11.0%), 362 (25.3%), and 246 (17.2%) were light, moderate, and heavy drinkers, respectively. The light, moderate, and heavy drinkers consumed 88.2 g, 181.2 g, and 418.1 g, respectively of ethanol a week on average. Older subjects had moderated their alcohol consumption significantly compared with the younger subjects ($p < 0.01$). In the 30s age group, 125 of the subjects (53.0%) were regular drinkers, compared with 246 (45.9%) in the 40s age group, 158 (36.5%) in the 50s age group, and 79 (34.8%) in the 60s age group.

The frontal lobe shrank with age ($p < 0.01$). In the 30s age group, only 18 (7.6%) had shrunken frontal lobes. In the 40s age group,

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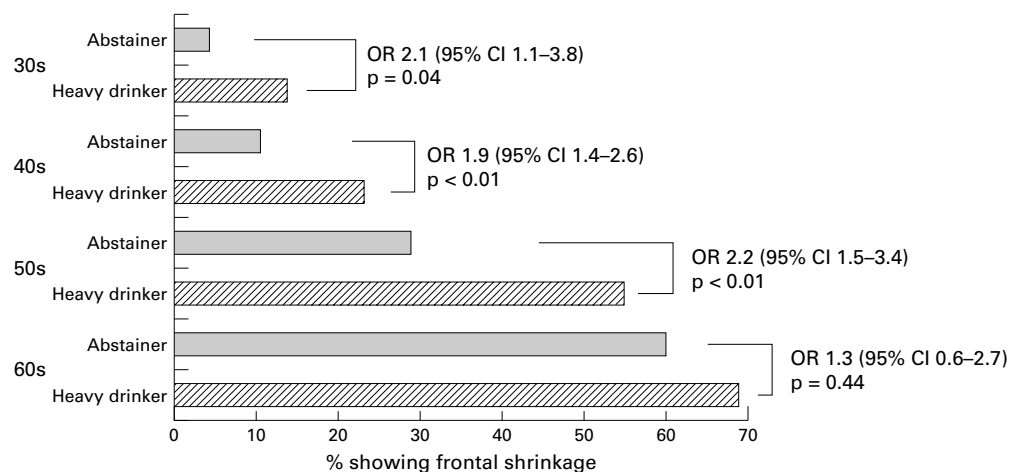


Figure 1 Alcohol consumption and brain atrophy. Heavy alcohol drinkers were at a significantly higher risk of brain shrinkage. Heavy alcohol consumption doubled the risk of brain shrinkage in the 30s to 50s age groups.

Table 1 Multivariate analysis by logistic regression

	OR	95%CI	p Value	CR (%)
Age	2.82	1.23–3.06	<0.01	31.0
Alcohol consumption	2.27	1.53–2.69	<0.01	11.3
Cigarette smoking	1.11	1.01–1.23	0.29	0
Hypertension	1.05	0.96–1.16	0.59	0
Diabetes mellitus	1.68	1.35–2.10	0.02	4.6
Hypercholesterolaemia	1.47	1.23–1.76	0.03	4.0

CR=Contributory rate.

84 (15.6%) of the subjects showed frontal lobe shrinkage, as did 164 (37.9%) in the 50s age group. More than half of the subjects (138, 60.8%) showed frontal lobe shrinkage in the 60s age group, and 46 subjects (20.3%) had moderately to severely shrunken frontal lobes.

Light to moderate alcohol consumption did not increase the rate of frontal lobe shrinkage, whereas heavy drinkers had significantly shrunken frontal lobes compared with abstainers. The odds ratio (OR) for moderate drinkers was 0.98 (95% confidence interval (95% CI) 0.73–1.33, $p=0.92$), whereas that for heavy drinkers was 1.80 (95% CI 1.32–2.46, $p<0.01$). Figure 1 shows the incidence in each age group of frontal shrinkage. In the 30s to 50s age groups, heavy consumption of alcohol doubled the risk for frontal lobe shrinkage (ORs 2.1 in the 30s, 1.9 in the 40s, and 2.2 in the 50s age groups, respectively). In the 60s age group, heavy drinkers showed a minimal and non-significant increase in risk (OR 1.3, $p=0.44$).

As shown in table 1, aging was the most powerful determinant of frontal lobe shrinkage after adjusting for other variables. The incidence was increased 2.8 times for each 10 years (95% CI 1.23–3.06, $p<0.01$). The contributory rate (CR) for aging was 31.0%. Alcohol consumption was the second most important independent determinant, with a CR of 11.3%. Compared with abstainers to moderate drinkers, heavy alcohol drinkers were at twice the risk for atrophy in the 30s to 50s age groups (OR 2.27, $p<0.01$).

Discussion

Many investigators have reported that heavy alcohol consumption accelerates brain shrinkage. However, most of these studies involved

alcohol dependent patients. It had not been definitively established whether social drinkers also have reduced brain volumes. Therefore, we evaluated the influences of chronic alcohol consumption on brain volumes in a consecutive 1432 non-alcoholic subjects.

We measured the frontal CSF space as the indicator for alcoholic brain shrinkage, because alcoholic brain damage is particularly known to affect the frontal lobes.^{2,3,5} After adjusting for other variables, aging remained the most powerful determinant of such brain shrinkage, which indicates that brain shrinkage is a physiological phenomenon, advancing with age. Light to moderate alcohol consumption seemed not to affect brain volume, whereas heavy alcohol consumption might exaggerate brain shrinkage in the non-alcoholic middle aged population. The contributory rate of alcohol consumption was 11.3%, and about a tenth of the atrophy might thus be attributable to the effect of alcohol. We failed to show this influence in the elderly group, possibly because of a ceiling effect. However, another possibility is that some of the former heavy alcohol consumers were classified into light to moderate drinkers, because older subjects tended to moderate their alcohol consumption. Further study is needed to evaluate the effects in an elderly population.

Alcoholic brain atrophy is known to be associated with reduced cerebral blood flow^{3,5,6} and glucose metabolism,⁷ impaired evoked potentials,³ and cognitive function⁴ and antisocial behaviour.^{2,5} As shown in this study, it is likely that about a tenth of the brain atrophy found in the normal population might be caused by excessive alcohol consumption. Chronic heavy alcohol consumption of more than 350 g ethanol a week should consequently be avoided. In our study, the moderate consumption of alcohol (average 181.2 g ethanol a week) did not affect frontal lobe volume. Fortunately, alcoholic brain damage is known to be, at least in part, reversible.^{4,8,9} After only a few months of abstinence, brain volume, cerebral blood flow, and neuropsychological impairment showed a gradual recovery.^{10,11}

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