Increased Stroke Risk Is Related to a Binge Drinking Habit

Laura Sundell, MD; Veikko Salomaa, MD, PhD; Erkki Vartiainen, MD, PhD; Kari Poikolainen, MD, PhD; Tiina Laatikainen, MD, PhD

Background and Purpose—Heavy alcohol consumption increases the risk for all strokes, whereas moderate regular alcohol consumption is associated with a lower risk for ischemic stroke. The purpose of this study was to evaluate the effect of different drinking patterns on stroke risk, independent of average alcohol intake.

Methods—A prospective cohort study of 15,965 Finnish men and women age 25 to 64 years who participated in a national risk factor survey and had no history of stroke at baseline were followed up for a 10-year period. The first stroke event during follow-up served as the outcome of interest (N=249 strokes). A binge drinking pattern was defined as consuming 6 or more drinks of the same alcoholic beverage in men or 4 or more drinks in women in 1 session. Cox proportional-hazards models were adjusted for average alcohol consumption, age, sex, hypertension, smoking, diabetes, body mass index, educational status, study area, study year, and history of myocardial infarction.

Results—Binge drinking was an independent risk factor for total and ischemic strokes. Compared with non–binge drinkers, the hazard ratio for total strokes among binge drinkers was 1.85 (95% CI, 1.35 to 2.54) after adjusting for average alcohol consumption, age, and sex; the association was diluted after adjustment for other risk factors. Compared with non–binge drinkers, the risk for ischemic stroke was 1.99 (95% CI, 1.39 to 2.87) among binge drinkers; the association remained statistically significant after adjustment for potential confounders.

Conclusions—This study found that a pattern of binge drinking is an independent risk factor for all strokes and ischemic stroke. (Stroke. 2008;39:3179-3184.)

Key Words: alcohol drinking ■ stroke ■ population ■ risk factors

The role of alcohol consumption in stroke risk has been widely studied. Many cohort and case-control studies have found an increased risk for hemorrhagic and ischemic strokes to be related to heavy alcohol consumption.1–3 A U- or J-shaped association has been observed between alcohol consumption and ischemic stroke, wherein light to moderate alcohol consumption has been shown to protect against ischemic stroke.1–4 The protective effect of light to moderate alcohol consumption against ischemic stroke has been assumed to be mediated through the effects of alcohol on lipid peroxidation, increased HDL cholesterol, alcohol-induced decreases in platelet aggregation, and increased fibrinolytic activity.

The association of alcohol consumption with hemorrhagic stroke is more linear than its association with ischemic stroke.1 Alcohol consumption increases the risk of both intracerebral (ICH) and subarachnoid (SAH) hemorrhages.4–7 Increased risk for ICH due to alcohol consumption has been attributed to alcohol-induced hypertension and impaired hemostasis. A transient increase in blood pressure during heavy alcohol intake and withdrawal may contribute to the rupture of small cerebral arteries or to an aneurysm, suggesting that episodic heavy drinking is a particular risk factor for SAH.6

The effects of binge drinking on health are suspected to be different from those of regular drinking. Although binge drinking is an independent risk factor for ischemic heart disease mortality,8,9 only a few studies have assessed the effects of different drinking patterns on stroke risk. Finnish researchers Hillbom and Kaste were the first to show that acute alcohol intoxication predisposes to ischemic strokes and aneurysmal SAHs within 24 hours.10,11 We have studied the long-term predictive value of binge drinking on stroke incidence.

A Swedish cohort study reported a higher risk of ischemic stroke among infrequent drinkers, occasional binge drinkers, and men who sometimes felt intoxicated than among lifelong abstainers.12 Regular light to moderate alcohol consumption was found to protect against ischemic stroke, but irregular or sporadic alcohol consumption reduced this benefit.2

The purpose of this prospective cohort study was to investigate the risk of total stroke, ischemic stroke, and hemorrhagic stroke in relation to alcohol drinking patterns and to determine whether a binge drinking pattern is a risk factor for stroke after adjusting for average alcohol consumption and other potential confounders.
Subjects and Methods

Subjects
The study population consisted of subjects examined in the national FINRISK surveys in the years 1987, 1992, and 1997. A stratified, random sample of people age 25 to 64 years living in the provinces of North Karelia and Kuopio, southwestern Finland, the Helsinki-Vantaa region, and the Oulu region was drawn from the Finnish population register. Stratification was performed by sex and by 10-year age group so that in the year 1987, the study sample comprised 500 men and women in each age group from the province of North Karelia and 250 men and women in each age group from the province of Kuopio and southwestern Finland. In 1992, the study sample comprised 250 men and women in each age group from the provinces of North Karelia and Kuopio, southwestern Finland, and the Helsinki-Vantaa region. In 1997, the study sample comprised 250 men and women in each 10-year age group from all 5 geographically defined areas, leading to a total sample size of 26,000 subjects. Of these subjects, 19,689 participated in the baseline survey. The response rate was 81% in 1987, 76% in 1992, and 72% in 1997, yielding an overall response rate of 76%. Those subjects (n=480) who participated in the baseline survey more than once were counted eligible only in the first study year. Subjects with previous stroke (n=318) were excluded from the analyses. The total number of eligible only in the first study year. Subjects with previous stroke participating in the baseline survey more than once were counted eligible only in the first study year. Subjects with previous stroke defined areas, leading to a total sample size of 26,000 subjects. Of these subjects, 19,689 participated in the baseline survey. The response rate was 81% in 1987, 76% in 1992, and 72% in 1997, yielding an overall response rate of 76%. Those subjects (n=480) who participated in the baseline survey more than once were counted eligible only in the first study year. Subjects with previous stroke (n=318) were excluded from the analyses. The total number of subjects was 18,891. The number of those subjects who reported alcohol consumption during the previous year was 16,143. Information on alcohol drinking patterns and on all other covariates was obtained for 15,256 subjects.

Baseline Survey
The baseline risk factor survey was performed with standardized methods according to the WHO MONICA protocol. A self-administered questionnaire with questions on health behavior, diseases, symptoms, socioeconomic factors, and medical history was mailed to the subjects with an invitation to attend a health examination. Laboratory tests and physical measurements were performed at the health examination site. Average alcohol consumption was assessed by a self-administered questionnaire regarding the usual quantity and frequency of consumption during the past 12 months before the study. Although changes in alcohol consumption may take place over time, it is generally assumed that such data on average alcohol consumption yield a reasonably good estimate of the long-term average alcohol intake. Consumption was assessed separately for beer, wine, and spirits with the following questions: “How often do you usually drink beer (similar questions for wine and spirits)?” and “How much beer do you usually drink at a time (similar questions for wine and spirits)?” Pure alcohol content per 1 standard drink of alcoholic beverage was estimated to be 12.5 g for beer, 12.0 g for wine, and 12.0 g for spirits. In this study, 1 standard drink was estimated to contain 33 cl for beer, 12 cl for wine, and 4 cl for spirits. Heavy drinkers were defined as those consuming >350 g/wk for men and >210 g/wk for women. Moderate drinkers were defined as those drinking below these limits but >230 g/wk for men and >150 g/wk for women, and the rest were defined as light drinkers. A binge drinking pattern was defined for men as consuming 6 or more drinks of the same beverage type on 1 occasion, and for women, 4 drinks. The widely used AUDIT screening questionnaire includes a question on consuming 6 or more drinks at a time, and the criterion for binge drinking for men was chosen to follow this definition. The questionnaire did not allow us to identify those subjects who consumed different types of alcoholic beverages on 1 occasion and reached a level of binge drinking only when the number of drinks of different beverages was summed. However, an earlier study in Finland showed that 83% of all binge drinkers reported spirit consumption and 52% reported consuming only spirits. Only 10% of binge drinkers reported consuming both spirits and beer, 11% spirits and wine, and 9% spirits, wine, and beer. Seventeen percent preferred beer or wine during heavy drinking occasions, and among them, 8% reported only beer consumption, 7% only wine consumption, and 2% wine and beer consumption. These results suggest that most binge drinkers consume only spirits on heavy drinking occasions.

Education was assessed with a question about the total years of full-time education. To control for the effect of birth year on educational status, we used the following education categories: low, medium, and high. These categories were calculated with birth year-specific tertiles of total years of education.

Physical measurements (height, weight, blood pressure) were performed by trained nurses at the examination site. Body mass index (BMI, kg/m²) was calculated as a measure of obesity. Blood pressure was measured from the right arm of the participant after 15 minutes of rest in a sitting position. Appearance of the Korotkoff sounds was recorded as the systolic blood pressure and the fifth phase as the diastolic blood pressure. The measurement was repeated after 1 minute, giving 2 systolic and 2 diastolic blood pressure values. The latter blood pressure value was used in this study. Participants were classified as having hypertension if their blood pressure was ≥140/90 mm Hg at baseline or if they were receiving antihypertensive drug therapy and had taken their medication within the previous week.

Smoking was assessed by structured self-administered questions about smoking habits. Based on their responses, participants were classified into 2 categories: current smokers and nonsmokers. Current smokers referred to those who had smoked regularly for at least 1 year and had smoked daily during the previous month. Other respondents were classified as nonsmokers (never-smokers, occasional smokers, ex-smokers). Diabetes was assessed on the basis of hospital discharge register data and self-reported diagnoses of diabetes. Subjects were classified as having diabetes if they reported either physician-diagnosed diabetes or impaired glucose tolerance or if they had been hospitalized with a diagnosis of diabetes (ie, the National Hospital Discharge Register indicated ICD-9 code 230 or ICD-10 codes E10 or E11). Causes of death and hospital discharges were classified with ICD-9 coding until the end of 1995 and with ICD-10 coding from the beginning of 1996 because use of these codes is standard in Finland.

Subjects were classified as having a history of myocardial infarction if on the questionnaire they reported myocardial infarction diagnosed by a physician or if they had been hospitalized with ICD-9 diagnosis codes 410, 411, or 412 or with ICD-10 codes I21, I22, I20.0, or I25.2 before the baseline examination. The hospitalizations were identified by record linkage of the study data with the National Hospital Discharge Register.

Criteria for previous stroke were either self-reported stroke diagnosed by a physician before the baseline examination or hospitalization with a diagnosis of stroke, ie, ICD-9 codes 430, 431, 436, 4330A, 4331A, 4339A, 4340A, 4341 or 4349A or ICD-10 codes I63, I60, I64, or I61, referring to ICH, ischemic stroke, and SAH diagnosed before the baseline study year. Subjects with previous strokes (n=318) were excluded from the statistical analyses.

Follow-Up
The study cohort was followed up for fatal and nonfatal strokes. Follow-up information was based on the Finnish National Hospital Discharge Register for nonfatal stroke events and on the National Causes of Death Register for fatal events. Survey data were linked to both the hospital discharge register and the causes of death register through identification numbers assigned to every resident of Finland. The follow-up period was 10 years if the baseline risk factor survey was conducted in 1987 or 1992 and 9 years if the baseline survey was carried out in 1997. Fatal and nonfatal stroke events were analyzed together. The coverage of the follow-up was 100% for stroke events that occurred in Finland during the follow-up period. In total, the follow-up period comprised 149,425 person-years.

The outcome event was defined as either the first fatal or nonfatal stroke. ICD codes 431 and I61 were classified as ICH. ICD codes 430 and I60 were classified as SAH. ICD codes 436, I63, 4340A, 4331A, 4339A, 4330A, 4341A, and 4349A were classified as ischemic stroke.
Statistical Methods

The differences in background characteristics of the study participants with and without a pattern of binge drinking were analyzed with t tests and χ² tests as appropriate. The Cox proportional-hazards model served to assess the independent contribution of a binge drinking pattern to the risk of stroke and to evaluate relative risks adjusted for average alcohol consumption and other risk factors. The main interest was to evaluate the contribution of the binge drinking habit to the risk of stroke among those subjects who consumed alcohol. Therefore, those who reported consuming alcohol in the previous year participated in the analyses, and unless otherwise indicated, the results were applied to that study sample. To evaluate the risk of the binge drinking habit on the entire population sample, we included those who quit consuming alcohol or had never consumed alcohol.

The analyses were carried out for all stroke events, ischemic stroke events, and hemorrhagic stroke events. Total stroke events (n=249) refer to ICHs (n=33), SAHs (n=37), and ischemic strokes (n=179) analyzed together. Of all strokes, 26 were fatal and 223 nonfatal. Because of the small number of hemorrhagic stroke events, ICHs and SAHs were combined to assess the risk of hemorrhagic stroke. Ischemic strokes were analyzed as a single group.

Long-term average alcohol consumption, age, study year, and BMI were included in the models as continuous variables. Because of a skewed distribution, logarithmic transformation of average alcohol consumption was used in the models. Sex, education, study area, hypertension, diabetes, smoking, and a history of coronary heart disease were included as categorical variables. The basic model included average alcohol consumption, sex, and age as covariates. Next, potential confounders were added to the model 1 by 1. Confounders included blood pressure, diabetes, smoking, study area, study year, education, and a history of myocardial infarction.

To assess the contribution of average alcohol consumption to the risk of stroke, Cox proportional-hazards models were performed by including logarithmically transformed alcohol consumption and other covariates, except the drinking pattern, into the models. To evaluate whether stroke risk for binge drinkers differed among men and women, we evaluated a possible interaction between drinking pattern and sex. We found no such interaction, and unless otherwise indicated, the results were applied to that study sample. To evaluate the contribution of average alcohol consumption to the risk of stroke among those subjects who consumed alcohol, the analysis was repeated for those subjects who had no heavy drinking pattern. The HR was 1.99 (95% CI, 1.39 to 2.87) after controlling for average alcohol consumption, sex, age, and other covariates. Next, potential confounders were added to the model 1 by 1. Confounders included blood pressure, diabetes, smoking, education, study area, and a history of myocardial infarction.

To evaluate the association between average alcohol consumption and the risk of stroke, Cox proportional-hazards models were performed by including logarithmically transformed alcohol consumption and other covariates, except the drinking pattern, into the models. To evaluate whether stroke risk for binge drinkers differed among men and women, we evaluated a possible interaction between drinking pattern and sex. We found no such interaction (P=0.3), so analyses were carried out for men and women together. The proportional-hazards assumption was examined graphically and found to be valid. All analyses were performed with the SAS statistical package, version 8.2 (SAS Inc, Cary, NC).

Results

The study cohort included 3558 subjects (1053 women and 2505 men) with a binge drinking pattern and 12,407 subjects without binge drinking pattern (Table 1). The subjects with a binge drinking pattern were younger, consumed on average significantly more alcohol, had a higher BMI, more often had hypertension, smoked notably more, and had a lower educational status than did subjects with no binge drinking pattern. Binge drinkers also had fewer myocardial infarctions. Drinking patterns differed slightly between geographic areas. Subjects living in the province of North Karelia had binge drinking patterns less frequently than did those living in other study areas. No difference was observed in diabetes incidence between the groups. Subjects with a binge drinking pattern had an increased risk for all strokes and ischemic stroke, whereas no such association was found between that drinking pattern and hemorrhagic stroke.

Compared with subjects with no binge drinking pattern, the hazard ratio (HR) for any stroke was 1.85 (95% CI, 1.35 to 2.54) among binge drinkers after adjusting for average alcohol consumption, sex, and age (Table 2). The HR remained significantly higher for binge drinkers after control-
Table 2. HRs (95% CIs) for Any Stroke Event Among Binge Drinkers Compared With Non–Binge Drinkers (N=15 256)

<table>
<thead>
<tr>
<th>Study area</th>
<th>Model 1 HR (95% CI)</th>
<th>Model 2 HR (95% CI)</th>
</tr>
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<tbody>
<tr>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Binge drinking pattern</td>
<td>1.85 (1.35–2.54)</td>
<td>1.39 (0.99–1.95)</td>
</tr>
<tr>
<td>Long-term average alcohol consumption*</td>
<td>1.00 (0.92–1.01)</td>
<td>0.98 (0.89–1.07)</td>
</tr>
<tr>
<td>Age (y)</td>
<td>1.10 (1.09–1.21)</td>
<td>1.09 (1.07–1.11)</td>
</tr>
<tr>
<td>Sex (0, 1)</td>
<td>0.52 (0.39–0.71)</td>
<td>0.61 (0.44–0.83)</td>
</tr>
<tr>
<td>Study area</td>
<td></td>
<td></td>
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<tr>
<td>North Karelia</td>
<td>1.00</td>
<td></td>
</tr>
<tr>
<td>Kuopio region</td>
<td>1.07 (0.76–1.53)</td>
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<tr>
<td>Southwestern Finland</td>
<td>0.90 (0.62–1.32)</td>
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<tr>
<td>Helsinki region</td>
<td>1.21 (0.79–1.84)</td>
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<tr>
<td>Oulu region</td>
<td>0.67 (0.32–1.39)</td>
<td></td>
</tr>
<tr>
<td>Hypertension (0, 1)</td>
<td>1.64 (1.20–2.24)</td>
<td></td>
</tr>
<tr>
<td>Smoking (0, 1)</td>
<td>2.21 (1.67–2.93)</td>
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<tr>
<td>Diabetes (0, 1)</td>
<td>2.01 (1.35–2.98)</td>
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<tr>
<td>BMI (kg/m2)</td>
<td>1.04 (1.01–1.08)</td>
<td></td>
</tr>
<tr>
<td>Level of education (1, 2, 3)</td>
<td>1.03 (0.87–1.22)</td>
<td></td>
</tr>
<tr>
<td>History of myocardial infarction (0, 1)</td>
<td>1.74 (0.94–1.01)</td>
<td></td>
</tr>
<tr>
<td>Study year</td>
<td>0.98 (0.94–1.01)</td>
<td></td>
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</tbody>
</table>

Table 3. HRs (95% CIs) for Ischemic Stroke Events Among Binge Drinkers Compared With Non–Binge Drinkers (N=15 256)

<table>
<thead>
<tr>
<th>Study area</th>
<th>Model 1 HR (95% CI)</th>
<th>Model 2 HR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Binge drinking pattern</td>
<td>1.99 (1.39–2.87)</td>
<td>1.56 (1.06–2.31)</td>
</tr>
<tr>
<td>Long-term average alcohol consumption*</td>
<td>1.02 (0.92–1.13)</td>
<td>1.00 (0.89–1.12)</td>
</tr>
<tr>
<td>Age (y)</td>
<td>1.13 (1.11–1.15)</td>
<td>1.12 (1.09–1.14)</td>
</tr>
<tr>
<td>Sex (0, 1)</td>
<td>0.49 (0.34–0.71)</td>
<td>0.57 (0.39–0.84)</td>
</tr>
<tr>
<td>Study area</td>
<td></td>
<td></td>
</tr>
<tr>
<td>North Karelia</td>
<td>1.00</td>
<td></td>
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<tr>
<td>Kuopio region</td>
<td>1.02 (0.67–1.550)</td>
<td></td>
</tr>
<tr>
<td>Southwestern Finland</td>
<td>0.79 (0.51–1.25)</td>
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<tr>
<td>Helsinki region</td>
<td>1.22 (0.76–1.98)</td>
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<tr>
<td>Oulu region</td>
<td>0.78 (0.35–1.72)</td>
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</tr>
<tr>
<td>Hypertension (0, 1)</td>
<td>1.34 (0.94–1.91)</td>
<td></td>
</tr>
<tr>
<td>Smoking (0, 1)</td>
<td>1.99 (1.43–2.78)</td>
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</tr>
<tr>
<td>Diabetes (0, 1)</td>
<td>2.04 (1.30–3.20)</td>
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<tr>
<td>BMI (kg/m2)</td>
<td>1.06 (1.03–1.10)</td>
<td></td>
</tr>
<tr>
<td>Level of education (1, 2, 3)</td>
<td>1.03 (0.84–1.23)</td>
<td></td>
</tr>
<tr>
<td>History of myocardial infarction (0, 1)</td>
<td>1.49 (0.91–2.48)</td>
<td></td>
</tr>
<tr>
<td>Study year</td>
<td>0.97 (0.93–1.02)</td>
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</tbody>
</table>

**Discussion**

In this prospective cohort study, we found that alcohol consumption with a binge drinking pattern was an independent risk factor for all strokes and ischemic stroke after adjusting for average alcohol consumption and other potential confounders. We found an increased risk for total stroke and ischemic stroke but not for hemorrhagic stroke. However, ischemic stroke comprised >70% of all strokes, and therefore the increased stroke risk relates mostly to ischemic stroke. Many previous studies have found an increased risk for hemorrhagic stroke among heavy drinkers. In this study, the number of cases of hemorrhagic stroke was small, and the statistical power was probably insufficient to detect any association between binge drinking and hemorrhagic stroke. Owing to the small number of hemorrhagic strokes, we combined ICHs and SAHs and analyzed them together. However, the natural cause and pathogenesis of ICH and SAH differ, and this may also dilute the association between binge drinking and hemorrhagic stroke. Furthermore, numerous cohort studies have found a positive association between average alcohol consumption and stroke. In this study, however, we found no association between average alcohol consumption and any type of stroke. This could be due to the small number of subjects reporting a heavy average alcohol consumption in our study sample. This was tested in a previous study with the same cohorts. Self-reported alcohol consumption was not associated with stroke risk, but excessive alcohol consumption as assessed by laboratory markers was strongly related to stroke risk. That study suggested that the serum γ-glutamyl transferase level predicts stroke risk better than does self-reported alcohol consumption.

**Mechanisms**

The role of hypertension as a risk factor for stroke, both ischemic and hemorrhagic, is well known. Hypertension is considered a major risk factor for all types of stroke, and other risk factors are thought to act in concert with hypertension. Alcohol consumption has been shown to increase blood pressure. Long-term habitual alcohol consumption results in a dose-dependent gradual elevation in blood pressure. However, drinking large amounts of alcohol on 1 occasion was found to increase blood pressure more than the same amount of alcohol consumed over a longer period. Acute alcohol consumption and withdrawal have been shown to cause a transient rise in systolic and diastolic blood pressure in normotensive subjects, suggesting that heavy episodic drinking has an adverse affect on blood pressure independent of the average intake level. In this study, we found that binge drinking increased ischemic stroke risk independently of the average alcohol consumption and other confounders, including hypertension. Because of the known causal pattern of association between alcohol consumption, blood pressure, and stroke, controlling for hypertension may obscure the relation between alcohol consumption and stroke. Therefore, we conducted the analyses with and without hypertension as a covariate. The HR was not attenuated after...
controlling for hypertension, suggesting that binge drinking may increase stroke risk through a mechanism other than hypertension. This finding is consistent with the findings of previous case-control studies, which found recent heavy alcohol consumption to be a risk factor for ICH and for ischemic stroke due to cardiogenic brain embolism after controlling for hypertension.

Heavy, chronic alcohol consumption is associated with cardiomyopathy, which is known to predispose to cardiac arrhythmias and heart failure, both of which increase the risk for embolic stroke. Furthermore, heavy episodic drinking, even of short duration, and the subsequent withdrawal period have been shown to trigger cardiac arrhythmias of both ventricular and supraventricular origin. These arrhythmias are often called “holiday heart.” The most common arrhythmias observed after heavy drinking include atrial fibrillation and flutter. If sustained, arrhythmias predispose to embolic stroke. Even though heavy alcohol drinking predisposes to cardiac arrhythmias, arrhythmias are often of relatively short duration and cease after cessation of drinking. Therefore, they may be considered a relatively minor risk factor for embolic stroke if found in an otherwise healthy heart without preceding heart failure or other abnormalities. A Finnish case-control study reported increased ischemic stroke risk among persons who consumed 151 to 300 g and >300 g of alcohol within the week preceding the onset of stroke. Consumption of >40 g of alcohol within the preceding 24 hours increased the risk for ischemic stroke due to cardiogenic embolism with a predisposing high-risk source, referring to atrial fibrillation, recent myocardial infarction together with an akinetic or hypokinetic left ventricular segment, dilated cardiomyopathy, left atrial thrombus, or patent foramen ovale. Furthermore, heavy alcohol intake within 24 hours before the onset of stroke was found to be a risk factor for ischemic stroke due to large-artery atherosclerosis. This was suspected to be the effect of acute heavy drinking on blood circulation, predisposing the dislodging of a local thrombus from the artery wall.

Light to moderate alcohol consumption has been related to lower rates of ischemic heart disease and ischemic stroke. The beneficial effect of moderate consumption is associated with a reduction in atherosclerosis. However, an angiographic study measured coronary occlusion in relation to the total amount of alcohol consumed, as well as to the pattern of alcohol intake. Regular drinkers showed significantly lower coronary occlusion scores than did occasional drinkers or nondrinkers, whereas a variable drinking pattern was associated with higher occlusion scores. Accelerated atherosclerosis associated with a pattern of occasional drinking has been linked to adverse lipoprotein composition, where the lowest total to HDL cholesterol ratio occurred among regular drinkers. Chronic heavy alcohol consumption is associated with a variety of blood clotting and platelet abnormalities. Effects on platelets and the coagulation cascade are considered a crucial mechanism for the precipitation of acute strokes by alcohol.

In alcoholic patients with no cirrhosis of the liver, the primary effect is on blood platelets, where both qualitative and quantitative abnormalities appear due to alcohol ingestion. These alterations cause a prolongation of the bleeding time and predispose to hemorrhagic complications, including hemorrhagic stroke. Alcoholics with cirrhosis have decreased levels of clotting factors in the presence of both platelet abnormalities and coagulation defects. In terms of ischemic stroke, acute alcohol consumption has been found to be associated with increased platelet aggregation and thromboxane formation. Furthermore, acute alcohol ingestion has been shown to decrease fibrinolytic activity, to increase factor VIII complex, and to shorten bleeding time. Also, the alcohol withdrawal period has been shown to be associated with alterations in platelet count and function. Reactive thrombocytosis and increased platelet aggregability during withdrawal may explain why drinkers with a habit of binge drinking have an increased risk for ischemic stroke.

**Strengths and Limitations**

Self-reported alcohol consumption is usually underestimated. Underestimation has been found to be a consequence mainly of underreported drinking frequencies, probably because people tend to forget light drinking sessions. Amounts of alcohol consumed at 1 time are reported more accurately. In the present study, we found no association between average alcohol consumption and stroke. This could be due to the underestimation of alcohol intake. Elevated y-glutamyl transferase levels indicating heavy alcohol consumption were found to be associated with increased stroke risk in a previous study of the same cohort. Another weakness is the lack of data on possible changes in alcohol intake over time.

In this study, heavy episodic drinkers, also known as binge drinkers, were defined as those men who consumed 6 or more drinks of the same alcoholic beverage type (beer, wine, or spirits) on 1 occasion and those women who consumed 4 or more drinks. Subjects who reached these limits by consuming drinks from more than 1 beverage type category on 1 occasion had to be classified as non–binge drinkers, because we did not have the information necessary to identify them separately. This probably diluted the observed associations between binge drinking and stroke, and the true associations might be stronger.

The risk for ischemic stroke among binge drinkers was attenuated only slightly after adjustment for average alcohol consumption, hypertension, age, sex, smoking, diabetes, obesity, study area, and education. Even though these are considered major risk factors for stroke, especially for ischemic stroke, other relevant confounders that we were unable to control for may exist. The use of oral contraceptives, hormone replacement therapy, and migraine are also considered risk factors for stroke. Their contribution to stroke is closely related to that of other risk factors (mostly hypertension and smoking) that we controlled for. However, some evidence suggests that migraine may be an independent risk factor stroke, but this evidence seems to apply mostly to subjects who experience migraine with aura and to oral contraceptive users.

The strengths of this study lie in the large general population cohort followed up for the first stroke event by record linkage to the National Hospital Discharge Register and the National Causes of Death Register. These registers have good coverage, and the accuracy of stroke diagnosis is high.
symptomatic stroke patients are hospitalized in Finland, and nearly all are diagnosed by routine computed tomography or magnetic resonance imaging examination, which is required for precise specification of stroke subtype. Our endpoint definition included symptomatic strokes, whereas subjects with mild, subclinical cases who were not hospitalized were not included. The large general population cohort, including information on alcohol consumption, drinking patterns, and other risk factors, allowed us to study the effect of a binge drinking habit on stroke risk after controlling for the effect of habitual alcohol consumption. Because of the heterogeneity of the nondrinking group, including lifelong teetotallers and abstainers who had quit drinking for health reasons, we compared drinkers with and without a binge drinking pattern.

Conclusions
This prospective cohort study showed that a binge drinking pattern is an independent risk factor for stroke; the highest risk was noted for ischemic stroke. A previous study found an increased risk for ischemic heart disease among binge drinkers. These results suggest that a binge drinking pattern adversely affects cardiovascular outcomes independently of average alcohol consumption.

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Disclosures
None.

References
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