Alcohol and Acute Ischemic Stroke Onset
The Stroke Onset Study
Elizabeth Mostofsky, MPH; Mary R. Burger, MD; Gottfried Schlaug, MD, PhD; Kenneth J. Mukamal, MD, MPH; Wayne D. Rosamond, PhD; Murray A. Mittleman, MD, DrPH

Background and Purpose—Previous research suggests that regular heavy alcohol consumption increases the risk for ischemic stroke, whereas frequent light to moderate alcohol intake may decrease the risk. However, the risk of ischemic stroke associated with transient exposure to alcohol remains unclear. In this study, we used a case–crossover approach to test the hypothesis that alcohol consumption affects the acute risk of ischemic stroke, to determine the length of time between alcohol intake and the onset of symptoms (induction time), and to examine whether the risk varies by the type of alcohol.

Methods—In this multicenter study, we interviewed 390 patients (209 men, 181 women) between January 2001 and November 2006 (median 3 days after stroke). Alcohol consumption in the hour before stroke symptoms was compared with its expected frequency based on the usual frequency of alcohol consumption over the prior year.

Results—Of the 390 patients, 248 (64%) reported alcohol consumption in the prior year, 104 within 24 hours and 14 within 1 hour of stroke onset. The relative risk of stroke in the hour after consuming alcohol was 2.3 (95% CI, 1.4 to 4.0; \( P = 0.002 \)). The relative risks were similar for different types of alcoholic beverages and when the sample was restricted to those who were not simultaneously exposed to other potential triggers.

Conclusions—The risk of stroke onset is transiently elevated in the hour after alcohol ingestion. (Stroke. 2010;41:1845-1849.)

Key Words: alcohol ■ case–crossover ■ cerebrovascular disorders ■ epidemiology ■ stroke
Institutional Review Boards at each participating center and informed consent was obtained from each patient. Interviewers used a structured questionnaire and asked patients to report the date and time of their first symptoms heralding their stroke. Patients were asked if they had consumed any alcoholic beverage in the year before their stroke. Patients who reported any alcohol consumption were also asked to report the last time that they had consumed an alcoholic beverage, their usual frequency of alcohol consumption over the prior year, the usual number of servings consumed each time they drank an alcoholic beverage, and the types of alcohol consumed (beer, wine, or liquor). A serving size of alcohol was defined as 12 ounces of beer, 4 ounces of wine, or 1.5 ounces of liquor straight or in a mixed drink. Patients were also asked to report the timing of their last exposure to other potential triggers and usual frequency of these factors over the prior year, including caffeine, cigarette smoking, marijuana, cocaine, stress, anger, and physical activity. Other information collected from the interview included medication use and symptoms on the day of the stroke.

Reliability and Validity of the Questionnaire
The test–retest reliability of the Stroke Onset Study questionnaire was assessed in 25 patients who were reinterviewed up to 6 days after their initial interview. The intraclass correlation for the usual frequency of alcohol consumption over the prior year was assessed in 25 patients who were reinterviewed up to 6 days after their initial interview. The test–retest reliability of the Stroke Onset Study questionnaire was 0.84, and there was perfect agreement for reporting of any alcohol consumption during the past year and during each of the first 2 hours before stroke onset (κ=1.0). In the subset of 181 subjects interviewed at Beth Israel Deaconess Medical Center who had high-density lipoprotein cholesterol levels measured at the time of hospitalization, the partial correlation between estimated alcohol consumption and high-density lipoprotein cholesterol level adjusting for sex, age, race, smoking, education, and physical activity was 0.35 (P=0.003), comparable to that found in the Second National Health and Nutrition Examination Survey.20

Study Design
The Stroke Onset Study used a case–crossover study design to assess the change in risk of acute ischemic stroke onset during a brief “hazard period” after consumption of alcohol. In the case–crossover design, control information for each patient is based on his or her own past exposure experience. Self-matching eliminates confounding by risk factors that are constant within individuals over the sampling period but differ between subjects. Alcohol use in the hazard period, the 1-hour period immediately before the onset of ischemic stroke symptoms, was compared with its expected frequency based on control data obtained from the patients. We used the usual frequency of alcohol consumption over the year before stroke to estimate its expected frequency in an average 1-hour period.

Statistical Analysis
Each patient in a case–crossover study forms his or her own stratum and thus is his or her own control.21,22 The ratio of the observed exposure frequency in the hazard period to the expected frequency was used to calculate estimates of the rate ratio as a measure of relative risk (RR). We multiplied the usual annual frequency of alcohol consumption by the hypothesized window of its physiological effect (1 hour in the primary analysis) to estimate the amount of person-time exposed to alcohol. The unexposed person-time was calculated by subtracting this value from the number of hours in 1 year. The data were analyzed using methods for cohort studies with sparse data in each stratum.

To estimate the length of time from alcohol consumption to the onset of ischemic stroke, RRs were calculated by comparing exposure within different hypothesized windows of its physiological effect with the estimated person-time exposed to alcohol in the previous year. We conducted stratified analyses to assess the effect of drink type (beer, wine, liquor), sex, age (<65 years of age versus ≥65 years of age), smoking status (current smokers versus nonsmok-ers), and stroke etiology and compared the RRs by means of a test for homogeneity.23

To evaluate whether potential triggers could account for the observed association, we conducted a sensitivity analysis excluding patients who engaged in other potentially triggering activities (ie, vigorous physical exertion and anger) in the hour before their stroke. In another sensitivity analysis, we used the number of drinks consumed in the week before the stroke as the control information. We were not able to examine the association between binge drinking and ischemic stroke, because only 1 person reported drinking ≥2 servings of alcohol in the hour before stroke onset. All reported probability values are 2-sided.

Results
The characteristics of the Stroke Onset Study patients are presented in the Table. Of the 390 patients with acute ischemic stroke, 248 (64%) reported that they had consumed alcohol in the prior year (wine, n=45; beer, n=29; liquor, n=142). Compared with nondrinkers, subjects who reported alcohol consumption were more likely to be male and to have ever smoked cigarettes. Among the 248 subjects who drank alcohol in the prior year, 47 (12%) reported drinking at least 1 serving of alcohol per day, 38 (10%) reported drinking at least once per week, and 163 (66%) reported drinking at least once per month. The median frequency of consumption among drinkers in the prior year

<table>
<thead>
<tr>
<th>Table. Clinical Characteristics of the Stroke Onset Study Population*</th>
<th>Alcohol Drinkers (n=248)</th>
<th>Nondrinkers (n=142)</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>68±14.5</td>
<td>69±13.7</td>
<td>0.11</td>
</tr>
<tr>
<td>Male</td>
<td>143 (58%)</td>
<td>66 (47%)</td>
<td>0.03</td>
</tr>
<tr>
<td>Smoking status</td>
<td></td>
<td></td>
<td>0.09</td>
</tr>
<tr>
<td>Never</td>
<td>72 (29%)</td>
<td>56 (39%)</td>
<td></td>
</tr>
<tr>
<td>Former</td>
<td>122 (49%)</td>
<td>63 (44%)</td>
<td></td>
</tr>
<tr>
<td>Current</td>
<td>54 (22%)</td>
<td>23 (16%)</td>
<td></td>
</tr>
<tr>
<td>Obesity (body mass index &gt;30 kg/m²)†</td>
<td>59 (24%)</td>
<td>31 (22%)</td>
<td>0.68</td>
</tr>
<tr>
<td>Diabetes</td>
<td>56 (23%)</td>
<td>39 (27%)</td>
<td>0.28</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>101 (41%)</td>
<td>45 (32%)</td>
<td>0.08</td>
</tr>
<tr>
<td>Hypertension</td>
<td>152 (61%)</td>
<td>100 (70%)</td>
<td>0.07</td>
</tr>
<tr>
<td>Atrial fibrillation</td>
<td>32 (13%)</td>
<td>22 (15%)</td>
<td>0.48</td>
</tr>
<tr>
<td>History of</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>MI</td>
<td>36 (15%)</td>
<td>14 (10%)</td>
<td>0.19</td>
</tr>
<tr>
<td>Stroke</td>
<td>41 (17%)</td>
<td>32 (23%)</td>
<td>0.14</td>
</tr>
<tr>
<td>TIA</td>
<td>29 (12%)</td>
<td>18 (13%)</td>
<td>0.77</td>
</tr>
<tr>
<td>Coronary revascularization</td>
<td>11 (8%)</td>
<td>31 (13%)</td>
<td>0.15</td>
</tr>
<tr>
<td>Carotid endarterectomy</td>
<td>2 (1%)</td>
<td>5 (2%)</td>
<td>0.66</td>
</tr>
<tr>
<td>Stroke etiology‡</td>
<td></td>
<td></td>
<td>0.31</td>
</tr>
<tr>
<td>Small vessel</td>
<td>67 (29%)</td>
<td>44 (37%)</td>
<td></td>
</tr>
<tr>
<td>Large vessel</td>
<td>46 (20%)</td>
<td>21 (18%)</td>
<td></td>
</tr>
<tr>
<td>Cardioembolic</td>
<td>57 (25%)</td>
<td>24 (20%)</td>
<td></td>
</tr>
<tr>
<td>Other/undetermined</td>
<td>60 (26%)</td>
<td>32 (26%)</td>
<td></td>
</tr>
</tbody>
</table>

*Mean±SD or no. (%).
†There was no data available on body mass index for 7 subjects.
‡At 1 of the centers, stroke etiology was not determined (n=39).
MI indicates myocardial infarction; TIA, transient ischemic attack.
was 2.0 times per week. Subjects reported that they typically drank small amounts each time (median=1 drink) and only 13 reported typically drinking >2 servings.

There were 169 subjects who reported exposure during the week before stroke, 104 subjects drank alcohol within 24 hours of stroke onset, and 14 drank within 1 hour of stroke onset. We found that within 1 hour after alcohol consumption, the risk of stroke onset was 2.3-fold higher (95% CI, 1.4 to 4.0; \( P=0.002 \)) compared with periods of nonuse. The RR was 1.6 (95% CI, 1.0 to 2.5; \( P=0.05 \)) in the second hour after drinking and returned to baseline thereafter (Figure 1). By 24 hours, there was a 30% lower risk (RR=0.7, 95% CI, 0.5 to 0.9; \( P=0.02 \)).

Among the 14 participants who consumed alcohol in the hour before stroke onset, 7 drank liquor, 5 drank beer, and 2 drank wine. The RR for alcohol consumption in the hour before stroke onset was strongest for liquor and weakest for wine, although the difference was not statistically significant (\( P \) for interaction=0.28; Figure 2). The RRs for alcohol consumption in the hour before stroke did not vary by sex, age, smoking status, or stroke etiology (\( P \) for interaction=0.62, 0.62, 0.12, and 0.43, respectively).

Among the 248 participants exposed to alcohol in the prior year, 63 participants were exposed to other potential triggers in the hour before stroke onset. Of the 14 people exposed to alcohol in the hour before stroke onset, 4 were also exposed to vigorous physical activity and 1 drank a caffeinated beverage. When we conducted an analysis excluding the 63 people exposed to any potential stroke trigger in the hour before stroke onset did not meaningfully alter the results.

The mean usual frequency of alcohol consumption during the past year was 4.42 times per week, similar to the mean frequency of reported alcohol consumption during the past week (4.23). In a sensitivity analysis using each patient’s reported frequency of consumption in the past week as the control information, the risk of ischemic stroke onset was 3.3-fold higher (95% CI, 1.2 to 9.3; \( P=0.03 \)) within 1 hour of consuming at least 1 serving of alcohol compared with periods with no alcohol intake. Excluding the 1 person who reported drinking >2 servings of alcohol in the 2 hours before stroke onset did not meaningfully alter the results.

Discussion

In this study, alcohol consumption was associated with a transient increased risk of ischemic stroke in the subsequent hour that was 2.3 times higher than the risk during periods with no alcohol consumption. This finding is consistent with previous research indicating an acute detrimental effect of alcohol consumption.\(^17,18\) The risk returned to baseline by 3 hours and there was a modestly lower risk by 24 hours.

Few studies have evaluated the role of alcohol as a trigger of ischemic\(^16–18,24\) and hemorrhagic stroke.\(^25\) For instance, Hillbom et al\(^18\) found that moderate (151 to 300 g) and heavy (>300 g) consumption of alcohol within the week before stroke onset is associated with a significantly higher risk of stroke with adjusted ORs of 3.6 (95% CI, 1.7 to 7.8) and 3.7 (95% CI, 1.6 to 8.7), respectively. Consistent with our data, Gorelick et al\(^24\) reported that after accounting for coexposures including smoking, there was no statistically significant increase in risk of ischemic stroke in the 24 hours after alcohol consumption.

Previous studies on the acute effects of alcohol consumption indicate that heavy alcohol intake is associated with impaired fibrinolysis,\(^2,3\) increased platelet activation,\(^4\) and increases in blood pressure and heart rate.\(^1\) Furthermore, heavy consumption may acutely lead to dehydration, further increasing the transient risk of stroke. However, such heavy consumption was rare and unlikely to explain our findings.

Even moderate drinking may have acutely adverse consequences. In a clinical trial of 8 healthy men, Hendriks and colleagues\(^7\) found that plasminogen activator inhibitor was significantly higher after 40 g of alcohol than water after 1, 3, and 5 hours but was not significantly different after 9 hours.

On the other hand, there is evidence that moderate drinking may provide transient health improvements.\(^5–9,11,12,26\) Short-term randomized trials indicate that moderate alcohol consumption may have beneficial effects through changes in flow-mediated vasodilatation\(^5,9\) within minutes to hours and improvements in lipid profile,\(^12\) inflammatory markers,\(^11\) soluble vascular adhesion molecules,\(^11\) and adipokines within weeks.

In contrast to the evidence on acute effects of alcohol consumption, there is consistent evidence that habitual heavy alcohol consumption is associated with an increased risk of ischemic\(^27,28\) and hemorrhagic stroke.\(^28–30\) However, the evidence regarding light to moderate consumption is mixed.
Although some studies reported no association with ischemic or hemorrhagic stroke,31–35 recent comprehensive reviews\(^28,36\) indicate a decreased risk of ischemic stroke and a higher risk of hemorrhagic stroke associated with light to moderate consumption.

Integrating the short-term effects observed here with other studies on alcohol use and long-term risk is difficult. Although speculative, it is possible that the transiently increased stroke risk from moderate alcohol consumption may be outweighed by the health benefits for the next 24 hours, but consuming multiple drinks at once may result in a sharp increase in acute risk with potential increased long-term risk as well. Mukamal and colleagues\(^27\) found that, compared with complete abstention from alcohol, light consumption (<1 drink daily) is not associated with stroke risk, moderate alcohol consumption (1 to 2 drinks daily) is protective, and intake of >2 drinks per day is associated with an increased risk of ischemic stroke. Our finding of an acute detrimental effect and a suggestion of a decreased risk over the 24-hour period after alcohol consumption seem consistent with these findings. For example, one may hypothesize that over time, individuals who drink large amounts infrequently primarily experience the acute detrimental effect, whereas subjects who drink small amounts frequently may still experience a transient increase in risk, but this may be offset in part by the subsequent reduction in risk that follows.

There are some limitations to our study. Because the case–crossover design uses subjects as their own controls, there can be no confounding by risk factors that are stable over time.\(^22\) Confounding by factors that change over time within individuals can occur. However, excluding subjects reporting other potential triggers in the hour before stroke onset did not materially alter the results. In an effort to minimize reporting bias, efforts were made to ensure the patient’s privacy during the interview. We used a standardized structured interview and patients were not informed of the duration of the hypothesized hazard period. Because most of the participants drank small amounts of alcohol in the hour before stroke onset, we could not examine the acute effects of different doses of alcohol. We had limited power to evaluate the effect of beverage type because few participants were exposed to each type. A larger study would help elucidate such effects. Finally, our results may not be generalizable to patients presenting with a severe or fatal stroke.

**Summary**

In conclusion, we found that the risk of ischemic stroke was transiently elevated for 2 hours after drinking as little as 1 serving of alcohol. The risk rapidly returned to baseline and was modestly lower by 24 hours. When examined in the context of long-term studies of alcohol consumption, the net clinical impact on ischemic stroke risk appears to depend on the frequency and quantity of alcohol consumption. Definitive evidence would require a long-term clinical trial, although such a trial would be logistically difficult and is unlikely to be carried out in the near future.

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**Disclosure**

None.

**References**


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背景和目的：早前的研究提示规律性大量饮酒会增加缺血性卒中的风险，而经常性的少量饮酒则能减少其风险。然而，一过性饮酒的缺血性卒中风险仍不明确。本研究采用病例交叉研究来检验饮酒对急性缺血性卒中风险的影响，以了解饮酒开始至症状出现的时程（诱发时间）并观察不同种类酒精饮料对卒中风险的影响。

方法：本多中心研究在2001年1月至2006年11月间，共询问390名患者（男性209人，女性181人，中位时间为卒中发病后3天）。基于既往前一年中通常的饮酒频率，将卒中症状出现前1小时内酒精的摄入与预期频率进行比较。

结果：390名患者中，248例（64%）在前一年内饮酒，104例在卒中发生前24小时内曾有饮酒，14人在起病前1小时内曾有饮酒。饮酒后1小时内卒中发生的相对危险为2.3（95%可信区间为1.4-4.0；P=0.002）。当严格控制样本于那些并未同时暴露于其他诱因的患者时，不同种类酒精饮料具有相类似的相对危险度。

结论：饮酒后1小时内卒中发生的危险会短暂地升高。

关键词：酒精，病例交叉研究，脑血管病，流行病学，卒中


本研究中，我们使用了病例交叉研究来验证饮酒会影响缺血性卒中的急性危险的假说，明确饮酒至症状出现（诱发时间）的时程，检验不同种类酒精饮料是否具有相类似的风险。

方法

研究人群

卒中起病研究（Stroke Onset Study, SOS）在三个医学中心（马萨诸塞州波士顿的Beth Israel Deaconess医学中心，北卡罗来纳州的北卡罗来纳大学医院，加拿大多伦多哥伦比亚省温哥华岛卫生局）中进行。在2001年1月至2006年11月期间，共纳入390例患者（男性209例，女性181例），在急性缺血性卒中发病后（卒中数天3天，范围在0-14天）进行访谈。研究人员通过回顾各医院卒中中心收治患者的入院日志和病历记录明确入组患者。另外，对急诊入院时符合新发急性卒中表现的患者进行筛查。卒中的病因学诊断使用TOAST分型[19]。

研究人员使用标准化简要表格记录患者的人口统计学数据、既往史和入院时实验室结果等数据。合格的参与者应符合神经科临床诊断或相关的影像学检查明确的急性缺血性卒中的诊断，语言为英语，在卒中事件前无痴呆。排除标准包括：无法明确卒中症状的起始时间；或因认知损害、不能回忆卒中发病时间、有失语或疾病危重无法完成持续30-45
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分钟访谈等原因而无法完成结构性访谈。所有中心中，43%的明确的缺血性卒中患者符合入选标准，其中83%患者同意入组，5.5%拒绝入组，12.5%患者在接触前已出院。研究方案经各分中心伦理委员会批准并获得每位患者的知情同意。

访谈人员使用结构性问卷询问患者出现症状的时间及起病前一天内是否饮酒。要求所有曾经有饮酒的患者提供末次酒精饮料摄入时间、前一年里饮酒的频率、每次饮酒量和饮酒种类(啤酒、葡萄酒或烈性酒)等信息。一份酒精量的定义为12盎司啤酒或4盎司葡萄酒或1.5盎司烈性酒的直接或混合饮用。患者同时被要求提供最后一次其他潜在诱因(包括咖啡因、烟草、大麻、可卡因、应激、愤怒和体力活动)的暴露时机及最近一年中这些诱因的暴露频率。其他在访谈中收集的信息还包括药物的使用和卒中当天的症状。

问卷的信度和效度

在25名患者中进行SOS问卷的重测信度评估，他们均在首次访谈后6天再次接受访谈。饮酒的既往频率在组内的相关性高度一致(r=0.84)，对最近一年的和卒中发生前2小时的饮酒报道也具有良好的一致性(k=1.0)。181例在Beth Israel Deaconess医学中心住院的患者接受了高密度脂蛋白胆固醇的检测，在校正性别、年龄、种族、吸烟史、教育、体力活动等因素后，饮酒的估计量与高密度脂蛋白水平存在相关性(r=0.35，P=0.003)，与第二次国家健康与营养检验调查一致[20]。

为了明确饮酒至出血性卒中发生的时间长度，RR值的计算比较了按生理作用使用不同假设时间窗的酒精暴露和估计所得的过去一年里暴露于酒精的人/时数。包装在卒中前一周内的饮酒量作为控制信息。我们无法明确酗酒与出血性卒中的关系，因为仅有1例患者在卒中发生前1小时饮酒2个单位。以上检验均为双侧检验。

结果

SOS中患者的特征见表。据报道，在390例急性出血性卒中患者中，有248例(64%)在近一年中
饮酒 ( 其中葡萄酒 45 例、啤酒 29 例、烈性酒 32 例、
大于一种 142 例)。与未饮酒者相比，饮酒者更倾向
于为男性和吸烟者。在 248 例发病前一年里饮酒的
患者中，47 例 (12%) 饮酒至少每日 ≥ 1 个单位，38
例 (10%) 至少每周一次，163 (66%) 至少每月一次。
患者最近一年中饮酒频率的中位数为每周 2.0
次，自诉每次饮酒量较少 ( 中位数为 1 个单位)，仅
13 例 饮酒量超过 2 个单位。

169 例患者在卒中前一周内有饮酒，104 例则在卒
中发生前 24 小时内饮酒，其中 1 周内的有 14 例。饮
酒后 1 小时内卒中发生的危险性是未饮酒者的 2.3 倍
(95% 可信区间 [95% CI]: 1.44-4.0 ; P=0.002)。饮酒后第 2 小时的卒
中发生的相对危险为 1.6 (95% CI: 1.0-2.5; P=0.05)，之
后相对危险回落至基线水平 (图 1)。在 24 小时，风险
则下降了 30% (RR=0.7, 95% CI: 0.5-0.9; P=0.02)。

在卒中发生前 1 小时内饮酒的 14 例中，7 人饮烈
性酒，5 人饮啤酒，2 人饮葡萄酒。卒中前 1 小时内饮
烈性酒的相对危险高于葡萄酒，但没有达到统计学意
义 ( 交互后的 P 值为 0.28 ; 图 2)。卒中前 1 小
时饮酒的相对危险与性别、年龄、吸烟史或卒中病因
无关 ( 交互后的 P 值分别为 0.62、0.62、0.12 和 0.43)。

在 248 例一年内饮酒的患者中，63 例在卒中发生
前 1 小时内暴露于其他潜在的诱因。在卒中发生前 1
小时内的饮酒的 14 例中，4 例曾进行剧烈的体力活
动，1 例摄入含有咖啡因的饮料。当排除了 1 小时内具
有其他潜在在卒中诱因的 63 例患者后，研究
结果与之前相似。

患者在最近的一年中平均饮酒频率为每周 4.42
次，与最近一周内的平均饮酒频率相一致 (4.23 次)。
用每位患者所报告的最近一周的平均饮酒频率为对
照所做的灵敏度分析，饮用至少 1 个单位酒后 1 小
时内的缺血性卒中发病风险是未饮酒期的 3.3 倍 (95%
CI, 1.2-9.3; P=0.03)。排除 1 例患者在卒中发生前 2
小时内摄入 2 个单位的酒精，分析结果无明显变化。

讨论
本研究发现饮酒后 1 小时内缺血性卒中的风险
将会短暂性地上升，其风险是未饮酒时的 2.3 倍。
本研究所显示的饮酒后的急性损害作用，与早前研
究的发现一致 [17,18]。卒中风险在饮酒 3 小时后回落
至基线水平，并于 24 小时到达低点。

个别研究进行了酒精作为缺血性 [16-18] 和出血
中前一周内中度 (151-300 g) 至重度 (>300 g) 饮酒是
卒中发生的显著高危因素，校正后的 OR 值为 3.6 (95%
CI, 1.7-7.8)，与本研究相一致。但 Gorelick 等 [24]
研究表明，在校正了包括吸烟在内的协同暴露因素后，
饮酒后 24 小时内的缺血性卒中风险未见明显升高。

早前对饮酒的急性作用的研究显示，大量的饮
的上升 [1] 相关。另外，大量饮酒会造成急性脱水，
并进一步增加了短期发生卒中的风险。然而，这样
大量的饮酒十分少见，也无法解释我们的研究结果。
即使中量饮酒也可能导致急性不良反应。在一项
对 8 例健康男性的研究中，Hendriks 等 [3] 发现相
对于饮水，在饮酒 40 g 后的第 1、3、5 小时内，血纤
溶酶原激活抑制物显著升高，9 小时后则无显著差异。

另一方面，有证据表明适度饮酒能短暂地改善
机体健康状态 [5-9,11,12,26]。短期随机试验显示，适度饮
酒可在饮酒数分钟至数小时引起血流介导的血管扩
张 [5-6]，并在饮酒后数周内改善血脂组成 [12]、炎性标

与饮酒的急性作用相反，一系列证据显示长期
大量饮酒与缺血性 [27,28] 和出血性 [28-30] 卒中的风险增
加相关。然而，少 - 中等量饮酒研究的相关证据并

图 1 饮酒后卒中起病时间。卒中前每 3 小时作为独立的危险期,
各小时的饮酒情况与对照期相比较。误差条线显示 95% 可信区间。
虚线显示基线的风险水平。

图 2 不同酒精饮料的相对卒中风险。误差条线显示 95% 可信区间。
虚线显示基线的风险水平。

Mostofsky et al Alcohol and Stroke Onset: The Stroke Onset Study