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
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 year before stroke onset was perfect agreement for reporting of any alcohol consumption (k=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, RRIs 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-

### Table. Clinical Characteristics of the Stroke Onset Study Population

<table>
<thead>
<tr>
<th></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 ≥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.

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=32; >1 type, 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...
was 2.0 times per week. Subjects reported that they typically drank small amounts each time (median was 2.0 times per week). Subjects reported that they typically drinking each hour was compared with that during the control period. The error bars indicate the 95% confidence limits. The dashed line indicates the baseline risk.

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 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.

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\(^{9,26} \) 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, recent comprehensive reviews 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 found that, compared with complete abstinence 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. 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 effects of pattern of alcohol intake on serum lipid levels in regular drinkers.

**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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**Disclosures**

None.

**References**


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Background and Objectives: Previous studies have suggested that moderate and heavy drinking increases the risk of ischemic stroke, while frequent moderate alcohol intake is protective. However, the risk of ischemic stroke secondary to acute consumption of alcohol is not well known. This study employed a case crossover design to test the hypothesis that alcohol consumption affects the risk of acute ischemic stroke, and to determine the timing of alcohol consumption relative to the onset of symptoms (onset time) and to examine the effect of different types of alcoholic beverages on stroke risk.

Methods: This multi-center study was conducted between January 2001 and November 2006. A total of 390 patients (209 male, 181 female, median time from stroke onset to interview was 3 days) were included. Based on the usual alcohol consumption frequency in the previous year, the alcohol consumption 1 hour before the onset of stroke symptoms was compared with the expected frequency.

Results: Among the 390 patients, 248 (64%) had drank alcohol in the previous year, 104 (26%) in the 24 hours before the stroke, and 14 (4%) in the 1 hour before the stroke. The relative risk of stroke within 1 hour of alcohol consumption per drink was 2.3 (95% CI 1.4-4.0; P = 0.002). When the data were controlled for other confounding factors, different types of alcoholic beverages had similar relative risks.

Conclusions: In this study, we used a case crossover design to test the hypothesis that alcohol consumption affects the risk of acute ischemic stroke, and to determine the timing of alcohol consumption relative to the onset of symptoms (onset time) and to examine the effect of different types of alcoholic beverages on stroke risk.

Keywords: Alcohol, case crossover study, stroke, epidemiology, ischemic stroke.

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The preliminary results were previously presented in an abstract at the 2006 International Stroke Conference.

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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年内是否饮酒。要求所有曾经有饮酒的患者提供末次酒精饮料摄入时间、前一年内饮酒的频率、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（0.84），对最近一年和卒中发生前2小时的饮酒报道也具有良好的一致性（κ=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（0.35，P=0.003），与第二次国家健康与营养检验调查[20]一致。

研究设计

SOS使用了病例交叉对照设计，来评估饮酒后的时间段（危险期）中，急性卒中发生风险的变化。在病例交叉设计中，每位患者自身既往所存在的暴露作为其对照信息。自身对照控制了患者在研究期间持续存在但又与其他个体不同的混杂因素，将患者危险期，即缺血性卒中症状发病前1小时的饮酒情况与基于控制数据中的预期频率相比较。我们使用卒中前一年中饮酒的频率来估计其平均1小时的频率。

统计分析

病例交叉研究中的每位患者形成了其各自独立的分层，并进行自身对照[21,22]。所观察到的危险期内的暴露频率与计算所得的预期频率的比率作为相对危险度（RR）。我们将既往每年饮酒频率乘以理论生理作用的时间窗（初步分析为1小时）来估计暴露于酒精数量的人/时数，与未暴露的人/时数在一年中减去。每层中所缺失数据的分析采用队列研究方法。为了明确饮酒至缺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。我们通过分层分析来评估不同饮酒频率、性别、年龄（<65岁与≥65岁相比较）、饮酒史（近期饮酒者与非饮酒者相比较）和卒中病因学，通过一致性检验来比较其RR值。

为了评估一些潜在的诱因是否与观察结果相关，我们将卒中前1小时内具有其他潜在诱因的（如剧烈体力活动或情绪激动）患者排除在外，进行了敏感性检验。在另一项敏感性检验中，我们将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缺血性卒中中的关系，因为仅有1例患者在卒中发生前1小时内饮酒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例急性缺血性卒中患者中，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 小时内的有 14 例。饮
酒后 1 小时内卒中发生的危险为无饮酒者的 2.3 倍
 (95% 可信区间 [CI] 1.4-4.0；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 小时内的饮酒中，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
烈性酒的相对危险较高，葡萄酒较低，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似 (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
将会暂时性地上升，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 [16-18,24]
和出血性卒中 [25] 的诱因的研究。如 Hillborn 等 [18]
发现卒中前一周内中度 (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
酒与纤溶损害 [2,3]、促进血小板聚集 [4] 及血压和心律
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的不良反应。在一
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 [5,8,11,26]。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 [56]，并在饮酒后数周内改善血脂组成 [22]、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 [27,29] 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
不一致。尽管一些研究报道了其与缺血性或出血性卒中并无相关性[31-35]，但近期系统综述[28,36]表明，少量中等量饮酒与缺血性卒中风险的下降及出血性卒中风险的上升相关。

将本研究中饮酒的短期作用与长期风险相整合是十分困难的。据推测，重度饮酒后24小时内也可能超过短期卒中风险的增加作用，但单次多种饮酒可能导致急性风险和潜在长期风险的同时增加。Mukamal等[27]发现，从不饮酒相比，少量饮酒（≤1单位/天）与卒中风险无关，中度饮酒（1-2单位/天）则有保护性，而每日2单位及以上的饮酒则与缺血性卒中风险的上升相关。我们发现饮酒的急性损害作用及在24小时后风险的下降与这些研究相一致。可以假设，偶尔大量饮酒者通常可能在一开始即发生酒精的急性损害作用，而少量规律饮酒者可能也同样发生卒中风险的短期升高，但这会得到之后一系列处的补偿。

本研究仍存在一些不足之处。由于病例交叉研究中的患者，以自身作为对照，则不会受到研究期间稳定的危险因素的影响。因此排除了那些在卒中前1小时内有短暂的升高。这一风险在之后迅速回降至基线水平。

总述

我们发现缺血性卒中风险在饮酒1单位后2小时内有短暂的升高。这一风险在之后迅速回落至基线水平，24小时后的卒中风险则有轻度降低。早些一饮酒的长期研究证实对缺血性卒中风险的综合作用则依赖于饮酒的频率和剂量。这也需要长期的临床试验来提供决定性的证据，但从逻辑上看，此类试验难以开展，在近期无法实现。