Original Contribution

Alcohol Consumption and Lower Extremity Arterial Disease among Older Adults

The Cardiovascular Health Study

Kenneth J. Mukamal¹, Margaret Kennedy², Mary Cushman³, Lewis H. Kuller⁴, Anne B. Newman⁴, Joseph Polak⁵, Michael H. Criqui⁶, and David S. Siscovick⁷,⁸

¹ Department of Medicine, Beth Israel Deaconess Medical Center, Boston, MA.
² Hemostasis Thrombosis Laboratory, Western Pennsylvania Hospital, Pittsburgh, PA.
³ Departments of Medicine and Pathology and Laboratory Medicine, College of Medicine, University of Vermont, Burlington, VT.
⁴ Department of Epidemiology, Graduate School of Public Health, University of Pittsburgh, Pittsburgh, PA.
⁵ Department of Radiology, New England Medical Center, Boston, MA.
⁶ Departments of Medicine and Family and Preventive Medicine, School of Medicine, University of California, San Diego, CA.
⁷ Department of Medicine, School of Medicine, University of Washington, Seattle, WA.
⁸ Department of Epidemiology, School of Public Health and Community Medicine, University of Washington, Seattle, WA.

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Few studies of the relation of alcohol intake to lower-extremity arterial disease (LEAD) have included clinical events and objective measurements repeated longitudinally. As part of the Cardiovascular Health Study, a study of older adults from four US communities, 5,635 participants reported their use of beer, wine, and spirits yearly. Incident LEAD was identified by hospitalization surveillance. Technicians measured ankle-brachial index 6 years apart in 2,298 participants. A total of 172 cases of LEAD were documented during a mean of 7.5 years of follow-up between 1989 and 1999. Compared with abstention, the multivariable-adjusted hazard ratios were 1.10 (95% confidence interval (CI): 0.71, 1.71) for <1 alcoholic drink per week, 0.56 (95% CI: 0.33, 0.95) for 1–13 drinks per week, and 1.02 (95% CI: 0.53, 1.97) for ≥14 drinks per week (p for quadratic trend = 0.04). These relations were consistent within strata of sex, age, and apolipoprotein E genotype, and neither lipids nor inflammatory markers appeared to be important intermediates. Change in ankle-brachial index showed a similar relation (p for quadratic trend = 0.01). Alcohol consumption of 1–13 drinks per week in older adults may be associated with lower risk of LEAD, but heavier drinking is not associated with lower risk.

alcohol drinking; peripheral vascular diseases

Abbreviations: ABI, ankle-brachial index; CHS, Cardiovascular Health Study; CI, confidence interval; HDL, high density lipoprotein; HR, hazard ratio; LEAD, lower-extremity arterial disease.

Considerable epidemiologic evidence suggests that moderate alcohol drinkers have lower risks of coronary heart disease than do abstainers or very light drinkers (1, 2). However, the association of alcohol consumption with lower-extremity arterial disease (LEAD) is less clear, despite the fact that LEAD affects approximately 10 million adults in the United States alone (3). Two prospective cohort studies have assessed alcohol use and risk of symptomatic LEAD; neither of them included objective measurements of LEAD (4, 5). Three other studies have assessed this relation cross-sectionally using ankle-brachial index (ABI) measurements, with inconsistent results (6–8). To our knowledge, no
previous studies have assessed alcohol intake and changes in objectively measured LEAD in a longitudinal fashion.

Kennedy et al. (9) recently identified predictors of 6-year decline in ABI among older adults participating in the Cardiovascular Health Study (CHS). In age-, sex-, and race-adjusted analyses, levels of high density lipoprotein (HDL) cholesterol tended to be inversely associated with risk, while fibrinogen levels were positively associated with risk (9). Given that alcohol use increases HDL cholesterol levels and lowers fibrinogen levels in clinical trials (10) and is associated with higher HDL cholesterol levels (11) and lower fibrinogen levels in the CHS (12), it seems reasonable to hypothesize that alcohol use could be inversely associated with deterioration in ABI through effects on these risk factors.

Given the paucity of information about alcohol use and LEAD, we examined the association of alcohol intake with several measures of LEAD in the CHS, a population-based cohort study of community-dwelling older adults.

MATERIALS AND METHODS

Study population and design

The CHS is a prospective study of 5,888 men and women aged 65 years or older who were randomly selected from Medicare eligibility lists in four communities in Pennsylvania, Maryland, North Carolina, and California. The recruitment procedures used have been described previously (13); 60 percent of contacted and eligible participants enrolled. Participants were not institutionalized or wheelchair-dependent, did not require a proxy for consent, were not under treatment for cancer at the time of enrollment, and were expected to remain in their respective regions for at least 3 years. In 1989–1990, 5,201 participants were recruited and examined (the original cohort); in 1992–1993, an additional 687 African-American participants were recruited and examined. For the current analyses, we excluded 23 participants with missing information on baseline alcohol use. The institutional review board at each participating center approved the study, and each participant gave informed consent.

The design and objectives of the CHS have been published previously (14). The baseline examination included administration of standardized medical history questionnaires, a physical examination, resting electrocardiography, and laboratory measurements. At yearly clinic visits, updated information on personal and medical history was obtained, blood pressure was measured, and a short physical performance battery was administered. The follow-up rate through the 1998–1999 examination for surviving participants was 94–95 percent.

Alcohol consumption

At the baseline visit and annually until 1999, participants reported their usual frequency of consumption of beer, wine, and liquor and the usual number of 12-ounce (355-ml) cans or bottles of beer, 6-ounce (177-ml) glasses of wine, and shots of liquor that they drank on each occasion. The full text of the CHS nutrition questionnaire is publicly available (15). For administrative reasons, alcohol consumption was not determined at the 1990–1991 CHS visit; it was assessed with a validated food frequency questionnaire (16), which provided similar information, in 1995–1996. At baseline, participants reported whether they had changed their pattern of alcohol consumption during the past 5 years and whether they had ever regularly consumed five or more drinks daily. Participants who reported abstention at baseline but responded “yes” to either of these questions were classified as former drinkers.

As in all previous analyses of the CHS cohort, we categorized participants into categories according to weekly ethanol consumption as follows: none, former, <1 drink weekly, 1–6 drinks weekly, 7–13 drinks weekly, and ≥14 drinks weekly. For regression analyses, longer-term abstainers who did not report former use served as the reference category.

Determination of peripheral arterial disease

Peripheral arterial disease was documented with multiple complementary methods in the CHS. First, LEAD status during follow-up was prospectively adjudicated by a central committee (17). The committee evaluated all hospitalizations with International Classification of Diseases, Ninth Revision, code 440.2 or 443.9 that had evidence of symptomatic disease in the medical record (the primary endpoint). LEAD status at baseline was defined as a history of surgical or percutaneous revascularization of the lower extremity or diagnosed claudication.

Second, both the original cohort and the African-American cohort underwent bilateral ABI assessment in 1992–1993 and again in 1998–1999 with a standardized protocol (18). ABI was calculated as the ratio of the average of two blood pressure measurements taken in the right arm, right leg, and left leg. The correlations for each duplicate blood pressure were as follows: left leg, 0.97; right leg, 0.97; and right arm, 0.95 (18). As previously described (9), we examined ABI decline in two ways: measured primarily as a binary variable (a decline of over 0.15 units (19) with a final ABI of 0.9 or less) and secondarily as a continuous variable, using the lower of the right and left ABIs in 1992–1993 as the index value.

Lastly, CHS participants were assessed for intermittent claudication at yearly intervals (except for 1995–1996 through 1997–1998) using the World Health Organization/Rose claudication questionnaire (20, 21). We limited use of this measure to sensitivity analyses, because it appears to reflect other causes of lower-extremity pain in this population (21). Details on the CHS protocol for adjudication and confirmation of other cardiovascular and cerebrovascular diseases (congestive heart failure, coronary heart disease, stroke, transient ischemic attack) by the CHS Cardiovascular Events Committee, including the algorithms used for classification, have been published previously (17, 22).

Other covariates

We defined hypertension as an average baseline seated blood pressure of ≥140 mmHg (systolic) or ≥90 mmHg
(diastolic) or a combination of self-reported hypertension and use of antihypertensive medication. Diabetes mellitus was defined as a fasting blood glucose level of ≥126 mg/dl or the use of antidiabetic medication. Field center staff directly measured weight and standing height yearly, and body mass index was calculated as measured weight in kilograms divided by standing height in meters squared. Leisure-time physical activity was assessed as a weighted sum of kilocalories expended in specific physical activities (23). We separated smoking into three categories (current, former, and never) and calculated pack-years of smoking at baseline based upon reported age at onset, age at cessation, and average use. We dichotomized baseline marital status (married vs. widowed, divorced, separated, or never married), annual household income (<$16,000 vs. ≥$16,000), and education (high school diploma or less vs. some vocational school or college). Depressive symptoms were assessed at baseline with the Center for Epidemiologic Studies Depression Scale (24). Apolipoprotein E genotype testing was performed as described (25).

Statistical analysis

For analyses of incident hospitalization for LEAD, we excluded 149 participants at baseline with confirmed LEAD and 81 with positive or missing information on claudication, leaving 5,635 participants eligible for analysis. Of these, 270 declined consent for genetic testing for cardiovascular diseases and 362 did not have the necessary DNA stored or were not successfully genotyped, yielding 5,003 participants with information on apolipoprotein E genotype.

Of the 4,732 participants with ABI measurements in 1992–1993, we excluded 788 participants with confirmed LEAD or with ABI measurements of ≤0.9 or >1.4 in 1992–1993, 1,601 participants who did not have a follow-up ABI measurement, and 45 who did not have a successful follow-up ABI measurement on the index side, as previously described (9). Thus, there were 2,298 participants eligible for analyses of change in ABI.

For analyses of incident LEAD, participants accrued person-time from the date of study entry to the date of their first study examination with confirmed hospitalized LEAD (the primary endpoint of interest), death, or the 1998–1999 clinic examination. In initial multivariate Cox proportional hazards regression analyses (model MV1), we simultaneously controlled for age, sex, race, and cigarette smoking (current and former smoking and pack-years of smoking). Based on published correlates of LEAD (9, 26) and alcohol use (11) in this cohort, we then performed analyses (model MV2) that additionally controlled for education, income, physical activity, marital status, depressive symptoms, diabetes, cardio- or cerebrovascular disease, and body mass index (with linear and quadratic terms). We also adjusted for hypertension and self-reported general health (potential intermediates) in sensitivity analyses. Additionally, we assessed baseline levels of HDL cholesterol, triglycerides, inflammatory markers (C-reactive protein, white blood cell count, fibrinogen), and fasting insulin and glucose as intermediates.

We tested the proportional hazards assumption using time-varying covariates (27) and found no violations. For tests of linear trend, we excluded former drinkers and treated mean intake within categories of alcohol use as a linear variable. For quadratic trend, we squared the linear trend variable after centering it at mean alcohol consumption.

We performed analyses using both baseline and updated measures of alcohol consumption, in which we prospectively assessed the risk of LEAD in yearly increments, based upon alcohol consumption derived from the preceding questionnaire (28). In these time-dependent analyses, we grouped participants who stopped drinking during follow-up with former drinkers at baseline and included cardiovascular disease, self-reported health, and hypertension as time-varying covariates. Where there were missing data on time-varying covariates, previous data were carried forward.

We conducted stratified analyses according to sex, median age, mean sex-specific physical activity, and the presence of an apolipoprotein E4 allele (which modifies several effects of alcohol consumption in this cohort) (11, 29–31). We tested for interaction in nested models with and without interaction terms.

We tested the association of alcohol intake in 1992–1993 (i.e., at the baseline ABI) with subsequent change in ABI in two ways. We primarily used logistic regression for the binary variable of marked decline and secondarily used generalized linear models with least-square means for change in ABI as a continuous variable. We adjusted for the covariates from Cox models and baseline ABI, as previously described (9), although patterns of association with alcohol intake were generally little changed by exclusion of baseline ABI as a covariate. To ensure that our results on ABI were not overly influenced by differential dropout over time, we also performed cross-sectional ABI analyses using the original CHS baseline data, excluding only those participants with missing data on ABI or confirmed LEAD.

RESULTS

Baseline characteristics

The characteristics of CHS participants according to baseline alcohol consumption are shown in table 1. Alcohol intake tended to be positively associated with male sex, cigarette smoking, income, and education and inversely associated with Black race and the prevalence of diabetes.

Average alcohol consumption and risk of LEAD hospitalization

Of the 5,635 eligible participants, 172 were hospitalized for LEAD during a mean of 7.5 years of follow-up. Table 2 shows results from age-, sex-, race-, and smoking-adjusted analyses and multivariable-adjusted analyses of the risk of hospitalized LEAD according to alcohol consumption. Analyses based upon alcohol intake updated yearly during follow-up showed a U-shaped relation between alcohol intake and risk, with approximately 45 percent lower risk among consumers of 1–13 drinks per week (for 1–6 and 7–13 drinks per week combined, hazard ratio (HR) = 0.56, 95 percent confidence interval (CI): 0.33, 0.95).

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There was no evidence of lower risk among consumers of <1 drink per week. In analyses using only baseline consumption, former drinkers were at higher risk than longer-term abstainers, but risk was not substantially higher or lower than that of longer-term abstainers in any other drinking category after multivariable adjustment. For both updated and baseline analyses, more extensively adjusted analyses yielded results closer to the null than did age-, sex-, race-, and smoking-adjusted analyses. When we evaluated smaller groups of covariates separately, sociodemographic factors (marital status, income, education) and clinical factors (diabetes, cardiovascular and cerebrovascular disease, body mass index) tended to account for similar degrees of confounding. In multivariable models that excluded cardiovascular and cerebrovascular disease as covariates, because their inclusion may represent overadjustment, the hazard ratio associated with 1–13 drinks per week was 0.54 (95 percent CI: 0.32, 0.92).

In sensitivity analyses, additional adjustment for low density lipoprotein cholesterol and serum creatinine among the 5,485 participants with available information yielded a hazard ratio for 1–13 drinks per week of 0.60 (95 percent CI: 0.35, 1.02). Likewise, our results did not change with additional adjustment for hypertension (for 1–13 drinks per week, HR = 0.57, 95 percent CI: 0.34, 0.96) or self-reported health status (HR = 0.58, 95 percent CI: 0.34, 0.98). We also found no substantial change in the lower risk associated with 1–13 drinks per week when participants with any previous coronary or cerebrovascular disease at baseline were excluded (HR = 0.55, 95 percent CI: 0.27, 1.12).

In stratified analyses (not shown), we found no consistent differences in the association of alcohol intake with risk of hospitalized LEAD among participants stratified by sex, age, physical activity, or apolipoprotein E genotype. Although only 59 never smokers developed hospitalized LEAD, the association with 1–13 drinks per week among these persons was also similar to that in the full CHS population (HR = 0.57, 95 percent CI: 0.19, 1.69). We could not conduct race-stratified analyses because only 30 African-American participants developed hospitalized LEAD.

We evaluated several biomarkers as possible intermediates of a relation between intake of 1–13 drinks per week and lower risk of clinical LEAD, including white blood cell count, fibrinogen, C-reactive protein, HDL cholesterol,

| TABLE 1. Characteristics of 5,635 participants who were free of lower-extremity arterial disease at baseline, according to usual alcohol consumption, Cardiovascular Health Study, 1989–1999* |
|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|
| Alcohol intake (no. of drinks/week) | Nondrinker | Former drinker | Usual alcohol consumption (no. of drinks/week) | <1 (n = 1,089) | 1–6 (n = 963) | 7–13 (n = 343) | ≥14 (n = 425) |
| Total | | | | 0.2 (0.2) | 2.0 (1.2) | 7.8 (0.9) | 20.7 (10.0) |
| Beer | 0 | 0 | 0.06 | 0.5 | 1.8 | 5.3 |
| Wine | 0 | 0 | 0.1 | 0.7 | 2.0 | 3.8 |
| Liquor | 0 | 0 | 0.07 | 0.8 | 2.9 | 11.6 |
| Age (years) | 73.3 (5.8) | 72.8 (5.7) | 72.6 (5.5) | 72.1 (5.4) | 72.9 (5.5) | 72.1 (5.0) |
| Female sex | 1,633 (71) | 206 (41) | 691 (63) | 448 (47) | 146 (43) | 165 (39) |
| Black race | 458 (20) | 128 (25) | 125 (11) | 114 (12) | 23 (7) | 37 (9) |
| Married | 1,434 (62) | 309 (62) | 716 (66) | 688 (72) | 252 (73) | 325 (77) |
| Current smoker | 197 (9) | 73 (15) | 126 (12) | 132 (14) | 37 (11) | 83 (20) |
| Pack-years of smoking | 12 (22) | 27 (32) | 17 (25) | 21 (26) | 23 (29) | 31 (32) |
| Hypertension | 1,427 (62) | 317 (63) | 609 (56) | 498 (52) | 162 (47) | 269 (63) |
| Body mass index† | 27.2 (5.2) | 26.8 (5.1) | 26.7 (4.6) | 26.4 (4.1) | 25.4 (3.6) | 25.6 (3.7) |
| Diabetes mellitus | 454 (20) | 132 (27) | 124 (11) | 109 (11) | 28 (8) | 45 (11) |
| CES-D‡ score (possible range, 0–30) | 5.0 (4.9) | 5.4 (4.9) | 4.7 (4.4) | 4.2 (4.3) | 3.6 (3.6) | 4.0 (4.2) |
| Leisure-time physical activity (kcal/day) | 1,543 (1,918) | 1,540 (1,941) | 1,758 (2,093) | 2,001 (2,159) | 1,995 (1,943) | 1,960 (2,205) |
| Some vocational school or college | 709 (31) | 165 (33) | 534 (49) | 546 (57) | 220 (65) | 255 (60) |
| Annual income >$16,000 | 995 (46) | 230 (48) | 619 (61) | 672 (74) | 238 (74) | 301 (75) |
| Low density lipoprotein cholesterol level (mg/dl) | 130 (37) | 125 (38) | 128 (34) | 126 (34) | 125 (34) | 123 (33) |
| Creatinine level (mg/dl) | 1.05 (0.39) | 1.11 (0.51) | 1.06 (0.40) | 1.07 (0.35) | 1.06 (0.27) | 1.06 (0.31) |

* Numbers and percentages are shown for categorical variables, and means and standard deviations are shown for continuous variables.
† Weight (kg)/height (m)².
‡ CES-D, Center for Epidemiologic Studies Depression Scale (24).
triglycerides, glucose, and insulin. Although all biomarkers except C-reactive protein were associated with risk in multivariable models when examined individually, their inclusion did not substantially change the risk associated with alcohol intake, whether they were included together (HR = 0.61, 95 percent CI: 0.36, 1.04) or separately.

In exploratory analyses, we also evaluated a composite outcome of hospitalized LEAD or reported claudication on the Rose questionnaire, which occurred in 348 participants. Updated alcohol intake was associated with the composite outcome in a U-shaped manner, but this association was weaker than was evident for the association with hospitalized LEAD alone and was substantially attenuated after multivariable adjustment (data not shown).

### Average alcohol consumption and ABI

Among the 2,298 eligible participants, ABI declined from a mean of 1.14 (standard deviation, 0.10) to 1.05 (standard deviation, 0.17), and 217 participants (9 percent) developed a marked decline (defined as a final ABI of ≤0.9 with a decline of >0.15 units). Table 3 shows the relation between alcohol consumption and marked ABI decline, using average alcohol intake at the time of the first ABI measurement. A U-shaped association was evident, with the lowest risk of ABI decline being seen among consumers of 7–13 drinks per week. Results using the mean 6-year change in ABI showed a similar U-shaped association ($p$ for quadratic trend = 0.06).

To evaluate whether these findings were driven by differential dropout over time, we performed similar cross-sectional analyses using the baseline ABI. After multivariable adjustment, mean ABI levels among abstainers, former drinkers, and consumers of <1, 1–6, 7–13, and ≥14 drinks per week were 1.07 (95 percent CI: 1.06, 1.08), 1.04 (95 percent CI: 1.03, 1.06), 1.07 (95 percent CI: 1.06, 1.08), 1.08 (95 percent CI: 1.07, 1.09), 1.09 (95 percent CI: 1.07, 1.11), and 1.06 (95 percent CI: 1.05, 1.08), respectively. Corresponding odds ratios for the prevalence of ABI ≤0.9 in comparison with abstainers were 1.58 (95 percent CI: 1.20, 2.08), 0.99 (95 percent CI: 0.78, 1.25), 0.60 (95 percent CI: 0.45, 0.80), 0.64 (95 percent CI: 0.42, 0.98), and 1.00 (95 percent CI: 0.72, 1.40).

### DISCUSSION

In this prospective cohort study of older adults, alcohol intake was associated with incidence of hospitalized LEAD in a U-shaped manner, with lower risk being observed only among consumers of 1–13 drinks per week. Decline in ABI over time tended to show a similar trend.

We incorporated two complementary measures of LEAD in our analyses, each of which had strengths and limitations. The primary clinical outcome relied on hospitalization-related diagnoses that were independently adjudicated and...
hence would identify advanced cases, albeit with high specificity. The difference between ABI measurements taken 6 years apart was used to provide an objective measure of LEAD regardless of symptoms, but this measure was subject to measurement error at both time points and was limited to participants who attended two examinations 6 years apart. Although none of these methods is ideal, their use together allowed us to examine a wide spectrum of LEAD. The similarity in our results obtained using hospitalized LEAD, ABI at baseline, and ABI decline also supports the robustness of our findings.

We found stronger but similar relations of moderate drinking with hospitalized LEAD in analyses that updated alcohol intake over time than in those limited to baseline alcohol use, suggesting that alcohol intake may have effects with a short latency period. The relation between alcohol intake and coronary heart disease is also stronger when updated measures are used (32). With the finding that baseline levels of lipids, inflammatory markers, and insulin did not appear to mediate this relation, our results may reflect the importance of short-term effects of alcohol intake on these biomarkers or on other risk factors not measured in the CHS, such as platelet aggregation (33) and activation (34) or endothelial cell dysfunction (35, 36).

The rich nature of the CHS study design allowed us to extend the work of other investigators in several ways. The Strong Heart Study and the Rotterdam Study both found inverse cross-sectional associations of moderate drinking with LEAD (defined as a low ABI) that tended to be strongest among women (7, 8), but other cross-sectional studies have not consistently confirmed this finding (6, 37), and none of these studies included change in ABI over time. In an analysis of symptomatic claudication in the Framingham Heart Study and the Framingham Offspring Study (5), there were hazard ratios of 0.67 (95 percent CI: 0.42, 0.99) associated with intake of 13–24 g/day among men and 0.44 (95 percent CI: 0.23, 0.80) associated with intake of 7–12 g/day among women, but former drinkers could not be separated from abstainers, and symptomatic claudication was the only measure of LEAD available. Physicians’ Health Study investigators (4) found a hazard ratio of 0.74 (95 percent CI: 0.57, 0.97) associated with intake of seven or more drinks per week, although only baseline information on alcohol consumption was available, and the composite outcome included only 66 cases of LEAD that required revascularization. Taken together with previous work, our findings support a U-shaped association of alcohol intake with LEAD similar to that reported for other manifestations of atherosclerotic vascular disease. Our study extended this relation to older adults, who have the greatest prevalence of LEAD.

Limitations of this study warrant discussion. Our results could have been influenced by differences between participants in factors other than alcohol consumption for which we did not control, such as diet. Likewise, although our results were unchanged by adjustment for baseline fasting glucose level, we had no information on duration of diabetes or degree of glycemic control, which could affect diabetic complications like LEAD. To have influenced our results, any uncontrolled confounder would have needed to be associated with both alcohol consumption and risk of LEAD and not closely correlated with the other covariates already in our multivariable models.

We relied on self-reported alcohol consumption in this study. However, older adults appear to report alcohol intake as accurately as younger persons (38), and alcohol use and HDL cholesterol have a correlation in the CHS (11) of the magnitude expected from other studies (16, 39).

Although the CHS is a population-based cohort study, participants were generally healthy, community-dwelling White and Black older adults. As a result, generalization of our findings to other populations, including Hispanic and Asian elders, must be made with an appropriate degree

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**TABLE 3. Risk of marked decline* in ankle-brachial index (ABI) according to usual alcohol consumption among 2,298 participants who had two ABI measurements taken 6 years apart, Cardiovascular Health Study, 1989–1999**

<table>
<thead>
<tr>
<th></th>
<th>Nondrinker (n = 789)</th>
<th>Former drinker (n = 385)</th>
<th>Usual alcohol consumption (no. of drinks/week)</th>
<th>p value†</th>
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<td>&lt;1 (n = 421)</td>
<td>1–6 (n = 398)</td>
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<td>No. with marked ABI decline</td>
<td>84</td>
<td>37</td>
<td>41</td>
<td>35</td>
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<tr>
<td>MV1 model‡</td>
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<td>HR§</td>
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<td>95% CI§</td>
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<td>MV2 model¶</td>
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<td>95% CI</td>
<td>0.63, 1.50</td>
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* A marked decline in ABI was defined as a decline of over 0.15 units, with a final ABI of 0.9 or less.
† p values were derived from tests of linear/quadratic trend in logistic regression models.
‡ The first multivariate (MV1) model adjusted for age, sex, race, and smoking (current, former, and pack-years).
§ HR, hazard ratio; CI, confidence interval.
¶ The second multivariate (MV2) model additionally adjusted for education, income, marital status, physical activity, depressive symptoms, body mass index, diabetes, and cardio- and cerebrovascular disease.
of caution. Similarly, too few participants had clinical LEAD or low ABI values during follow-up to establish a relation between alcohol intake and LEAD progression among those with established disease.

Because of the relatively low levels of alcohol intake among CHS participants (and in other older populations), it is difficult to determine the dose-response relations with precision, especially at high levels of intake. Intake of individual beverage types was also too limited to allow stable analyses; Djousse et al. (5) found lower risks of symptomatic LEAD with both beer and wine intake. Finally, no measure of binge drinking was available in the CHS, although binge-drinking rates are generally low in older adults.

In summary, consumption of 1–13 alcoholic drinks per week was associated with lower risk of hospitalized LEAD in this population of older adults, with a similar trend for risk of decline in ABI over time, but heavier drinking was not associated with lower risk. These associations must be placed in the context of the many potentially detrimental effects of alcohol drinking among older adults (40). Given that over 10 percent of older adults have evidence of LEAD, further studies are still needed to outline the effects of alcohol consumption among those with clinically recognized disease.

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Conflict of interest: none declared.

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