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Alcohol intake and brain structure in a multiethnic elderly cohort

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SUMMARY

Background & aims: Evidence suggests that consuming light-to-moderate amounts of alcohol reduces the risk of dementia and is associated better cognitive function and less cardiovascular disease, relative to those consuming no or heavy alcohol. There are only minimal data on the association between alcohol and brain magnetic resonance imaging (MRI) markers. This study aimed to examine the association between alcohol and brain structure measured with MRI.

Methods: In this cross-sectional study, high-resolution structural MRI was collected on 589 multi-ethnic community residents of New York aged ≥65 with available alcohol intake assessments via a food frequency questionnaire. Total brain volume (TBV), white matter hyperintensity volume (WMHV), and presence of infarcts were derived from MRI scans with established methods. We examined the association of alcohol intake with these imaging markers using regression models adjusted for demographic, clinical, and vascular risk factors.

Results: Compared to non-drinking, light-to-moderate total alcohol ($b = 0.007$, $p = 0.04$) or wine ($b = 0.008$, $p = 0.05$) intake, but not beer or liquor intake, was associated with larger TBV. Further analysis showed a dose–response association between alcohol (p-trend = 0.03) or wine (p-trend = 0.006) and TBV. Overall, alcohol intake was not associated with WMHV or brain infarcts.

Conclusions: Our study suggests that among older adults in the community, light-to-moderate alcohol intake, in particular wine, is associated with larger TBV. These findings suggest that light to moderate alcohol consumption is potentially beneficial for brain aging, but replication is needed.

1. Introduction

A large amount of evidence from longitudinal epidemiological studies suggests that people consuming light-to-moderate amounts of alcohol have a lower risk of dementia[1–3] or better cognitive function,[3,4] and lower risk of ischemic stroke[5] than do persons who either abstain from or consume heavy amounts of alcohol. It is currently unclear which biological mechanisms could be involved in these associations.

A number of neuroimaging markers, including global and regional brain volume,[6] white matter hyperintensity volume (WMHV),[7] and brain infarcts,[8] have been related to cognitive functioning, and risk of dementia and stroke. Neuroimaging markers may reflect different biological changes. For instance, decreased relative brain volume represents brain atrophy, and increased WMHV and presence of brain infarcts represent small and larger vessel cerebrovascular disease, respectively. Therefore, examining the associations of alcohol intake with neuroimaging markers may

Non-standard abbreviations: APOE, Apolipoprotein; MRI, magnetic resonance imaging; SFFQ, semi-quantitative food frequency questionnaire; TBV, total brain volume; TCV, total cranial volume; WHICAP, Washington Heights/Hamilton Heights Inwood Columbia Aging Project; WMHV, white matter hyperintensity volume.

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help clarify potential underlying mechanisms for the apparent association between alcohol intake and a protection against neurological or vascular diseases.

The few studies that have examined the association between alcohol consumption and brain MRI markers at a population level had mixed results.[9–16] No previous study has examined the effect of alcohol on brain imaging findings among diverse populations that include Hispanics. In addition, different alcoholic beverages may share the same alcohol content, but they also contain different non-alcoholic ingredients such as anti-oxidant polyphenols, which can be found in wine but not in beer or liquor.[17] Thus, wine, beer, and liquor may be protective or may confer risk for suboptimal brain aging via different brain markers. However, the independent role of beer, wine, and liquor has rarely been assessed. In an attempt to fill some of these gaps, we sought to examine the association between alcohol (total alcohol, beer, wine, and liquor) intake and brain volume, WMHV, and brain infarcts among participants of the Washington Heights/Hamilton Heights Inwood Columbia Aging Project (WHICAP), a community-based study of multiethnic, elderly (65 years or older) people in New York. Based on previous reports of a protective cognitive and vascular effect of moderate alcohol consumption, we hypothesized that light-to-moderate alcohol consumption would be associated with markers of healthier brain aging.[1–5]

2. Materials and methods

2.1. Study participants

The WHICAP participants were identified (via ethnicity and age stratification processes) from a probability sample of Medicare beneficiaries aged 65 or older, residing in northern Manhattan.[18] The initial sample for this study included 2776 participants of the ongoing WHICAP II cohort. Briefly, at entry, a physician elicited each participant's medical and neurological history, and conducted a standardized physical and neurological examination. Each participant also underwent a structured in-person interview including an assessment of health and function and a neuropsychological battery.[18] Participants were followed at intervals of approximately 1.5 years, repeating the baseline examination and consensus diagnosis. The diagnosis of any type of dementia or its absence was based on standard research criteria, using all available information (except the MRI results) at a consensus conference of physicians, neurologists, neuropsychologists and psychiatrists. The diagnosis was made blind to alcohol consumption information. The Columbia University Institutional Review Board has reviewed and approved this project. All individuals provided written informed consent.

In 2004, we began systematic collection of high-resolution neuroimaging data on ongoing dementia-free WHICAP II participants.[19,20] A total of 769 WHICAP participants received MRI scans.[20] Fifty-two subjects met diagnostic criteria for dementia at the clinical evaluation closest to neuroimaging were further excluded. Of the remaining 717 subjects, 114 had no alcohol assessments available at the MRI visit. The 603 subjects with alcohol assessments were demographically and clinically similar (data not shown) to those 114 subjects with missing alcohol assessment.

2.2. MRI protocol

Scan acquisition was performed on a 1.5T Philips Intera scanner at Columbia University Medical Center and transferred electronically to the University of California at Davis for morphometric analysis.[20]

User-operated image analysis was performed on a workstation (Ultra 5; Sun Microsystems, Santa Clara, California) using the Quantium 6.2 software package (Sun Microsystems). Subject identifying information was not available to the operator. We have previously reported the methods used to derive the two morphologic variables, i.e., total brain volume (TBV) and WMHV.[20] TBV was the sum of voxels designated as brain volume from the segmentation process. WMHV was calculated as the sum of voxels 3.5 SD or greater above the mean intensity value of the image and multiplied by voxel dimensions and section thickness. TBV was adjusted for total cranial volume (TCV), and the ratio of TBV to TCV [i.e. relative TBV (rTBV) = TBV/TCV × 100] was used in the analyses. Similarly, WMHV was adjusted for TCV, and relative WMHV (rWMHV) was used in the analyses.

The presence or absence of brain infarction on MRI was determined using all available images, as previously described.[19] Only lesions ≥3 mm qualified for consideration as brain infarcts. Two raters determined the presence of cerebral infarction on MRI. Previously published κ values for agreement among raters have been generally good, ranging from 0.73 to 0.90.[19]

2.3. Alcohol consumption

Information about average alcohol consumption (beer, wine, or liquor) over the prior year was obtained using the 61-item version of Willett's semi-quantitative food frequency questionnaire (SFFQ) (Channing Laboratory, Cambridge, MA), administered by trained interviewers in English or Spanish. Participants reported one of the following combinations of frequency and amount of alcohol consumption: <1 drink/month, 1–3 drinks/month, 1 drink/week, 2–4 drinks/week, 5–6 drinks/week, one drink/day, 2–3 drinks/day, 4–5 drinks/day, and ≥6 drinks/day.[1] One drink was 12-oz beer, 4-oz wine, or 1.5-oz liquor, each containing approximately 10 g of alcohol. These serving frequencies were converted to monthly consumption of alcohol drinks. Because of the U-shape relationship between alcohol intake and the neurological outcomes[1–5] and the small number of heavy drinkers in this study population, participants were classified into three categories: none (currently not drinking), light-to-moderate (>0 and ≤30 drinks/month for women and >0 and ≤60 drinks/month for men), and heavy (>30 and >60 drinks/month for women and men, respectively) drinkers according to the Dietary *Guidelines for Americans 2010* definition of moderate drinking (up to 1 drink per day for women and up to 2 drinks per day for men).[21] A total of 409, 180 and 14 subjects reported no, light-to-moderate, and heavy drinking of alcohol, respectively. Considering the small number of heavy-drinkers, and the potential difference between non-drinkers and heavy-drinkers, we included in the analysis only the 589 non-drinkers and light-to-moderate drinkers. The measurements of alcohol intake between two SFFQs administered 2 months apart were not significantly different, suggesting a satisfying reliability of alcohol measurement by SFFQ in the current study population.[1]

2.4. Covariates

Age (years), education (years), caloric intake (kcal), and body mass index (BMI; weight in kilograms divided by height in square meters [kg/m^2]) were used as continuous variables. Participants were assigned to one of four groups: African American (Black non-Hispanic), Hispanic, White (non-Hispanic) or Other based on self-report using the format of the 1990 US census. Ethnicity was used as a dummy variable with non-Hispanic White and Other as the reference. Sex was used as a dichotomous variable with male as the reference. Apolipoprotein (APOE) genotype was used dichotomously: absence of ε4 allele (as reference) vs. presence of either 1 or 2 ε4 alleles. Smoking status was used dichotomously: ever smoke vs. never smoked, with never smoked as the reference group. History of diabetes, hypertension, heart disease, or clinical stroke