Association of moderate alcohol intake with in vivo amyloid-beta deposition in human brain: A cross-sectional study

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Abstract

Background

An emerging body of literature has indicated that moderate alcohol intake may be protective against Alzheimer disease (AD) dementia. However, little information is available regarding whether moderate alcohol intake is related to reductions in amyloid-beta (Aβ) deposition, or is protective via amyloid-independent mechanisms in the living human brain. Here we examined the associations of moderate alcohol intake with in vivo AD pathologies, including cerebral Aβ deposition, neurodegeneration of AD-signature regions, and cerebral white matter hyperintensities (WMHs) in the living human brain.

Methods and findings

The present study was part of the Korean Brain Aging Study for Early Diagnosis and Prediction of Alzheimer's Disease (KBASE), an ongoing prospective cohort study that started in 2014. As of November 2016, 414 community-dwelling individuals with neither dementia nor alcohol-related disorders (280 cognitively normal [CN] individuals and 134 individuals with mild cognitive impairment [MCI]) between 56 and 90 years of age (mean age 70.9 years ± standard deviation 7.8; male, n [%] = 180 [43.5]) were recruited from 4 sites (i.e., 2 university hospitals and 2 public centers for dementia prevention and management) around Seoul, South Korea. All the participants underwent comprehensive clinical assessments comprising lifetime and current histories of alcohol intake and multimodal brain imaging, including [11 C] Pittsburgh compound B positron emission tomography (PET), [18 F] fluorodeoxyglucose (FDG) PET, and magnetic resonance imaging (MRI) scans. Lifetime and current alcohol intake were categorized as follows: no drinking, <1 standard drink (SD)/week, 1–13 SDs/week, and 14+ SDs/week. A moderate lifetime alcohol intake (1–13 SDs/week) was significantly associated with a lower Aβ positivity rate compared to the no drinking group, even after controlling for potential confounders (odds ratio 0.341, 95%

confidence interval 0.163–0.714, p = 0.004). In contrast, current alcohol intake was not associated with amyloid deposition. Additionally, alcohol intake was not related to neurodegeneration of AD-signature regions or cerebral WMH volume. The present study had some limitations in that it had a cross-sectional design and depended on retrospective recall for alcohol drinking history.

Conclusions

In this study, we observed in middle- and old-aged individuals with neither dementia nor alcohol-related disorders that moderate lifetime alcohol intake was associated with lower cerebral Aβ deposition compared to a lifetime history of not drinking. Moderate lifetime alcohol intake may have a beneficial influence on AD by reducing pathological amyloid deposition rather than amyloid-independent neurodegeneration or cerebrovascular injury.