A long-term Australian study has revealed that drinking higher amounts of coffee may reduce the risk of developing Alzheimer’s disease (AD) ([1]).

AD is a neurodegenerative disease characterized by progressive impairment of learning, memory and other cognitive deficits, with extracellular deposition of Aβ-amyloid protein within the brain leading to neuroinflammation, synaptic loss and neuronal death ([2]).

Coffee is one of the most popular beverages consumed worldwide, due to its stimulating effects on the central nervous system as well as its taste and aroma. Epidemiological studies suggest coffee has beneficial effects on various conditions including stroke ([3]), dementia ([3]), heart failure ([4]), cancers ([5]), diabetes ([6]), and Parkinson’s disease ([7]). Coffee consumption has previously been linked to decreased risk of mild cognitive impairment (MCI) and AD ([8],[9],[10]), however, this is the first study investigating the relationship of coffee intake to rates of decline in multiple cognitive domains.

The study investigated the relationship between self-reported habitual coffee intake, and cognitive decline in 227 cognitively normal older adults (average age 69.7 years at baseline) over 126 months (10.5 years). Cognitive decline was assessed using a comprehensive neuropsychological battery. In a subset of individuals, the relationship between habitual coffee intake and cerebral Aβ-amyloid accumulation and brain volumes was also assessed.

Results showed higher coffee consumption was associated with slower cognitive decline, specifically in the attention and executive function domains, which includes planning and self-control. Higher coffee consumption was linked with a lower risk of transitioning from cognitively normal to MCI or AD status over 126 months.

In the neuroimaging subset, higher coffee intake was associated with slower cerebral Aβ-amyloid accumulation, as well as lower risk of progressing to “moderate,” “high,” or “very high” Aβ-amyloid
burden status over the same time period. Coffee consumption was not, however, associated with rates of grey matter, white matter or hippocampal volume atrophy.

The observed effect size estimates of the study suggest that increasing intake from one to two cups of homemade coffee (240g) per day could potentially provide up to 8% decrease in executive function decline over 18 months, and up to 5% decrease in cerebral Aβ-amyloid accumulation. Further longitudinal observational and intervention studies are required to substantiate this suggestion. A maximum number of cups per day that provided a beneficial effect was not able to be established from the current study.

Coffee contains a range of bioactive compounds, including caffeine, chlorogenic acid, polyphenols and small amounts of vitamins and minerals ([11]). While caffeine has been linked to the results in the current study, animal model studies of AD provide preliminary evidence to suggest the observed benefits are not due to caffeine alone. Crude caffeine, a by-product of the decaffeination process, is as effective as caffeine in partially preventing memory impairment in AD mice ([12]). Other coffee components such as cafestol, kahweol and eicosanoyl-5-hydroxytryptamide also affected cognitive impairment in animals in various studies ([13],[14],[15]).

The blocking of adenosine receptors by caffeine, leading to a decrease in Aβ-amyloid in the brain, and subsequent reduction of tau hyperphosphorylation is a potential mechanism that warrants further investigation.

This study adds to the growing evidence supporting the hypothesis that coffee intake may be a protective factor against AD. A 2016 meta-analysis of nine prospective cohort studies found that drinking 1-2 cups of coffee per day was associated with a lower incidence of cognitive disorders (i.e., cognitive decline, cognitive impairment, AD, and all-cause dementia) compared with less than one cup. Studies ranged in follow-up from 1.3 to 28 years ([8]).

A recent cross-sectional analysis found self-reported lifetime intake of > 2 cups of coffee per day was associated with lower rates of “Aβ positivity” (presence of significant brain Aβ-amyloid), compared to < 2 cups per day (n=411 cognitively normal older adults). However, in this cohort, current coffee intake was not related to “Aβ positivity”, and neither current nor lifetime intake was related to brain volume or cerebral cortical thickness ([16]), consistent with findings from the current study ([1]).

Limitations

Known potential confounding factors were adjusted for in the study yet there is a possibility of residual confounding factors that were not measured. Due to the nature of the study, there is the possibility of measurement error or recall bias concerning dietary data. Coffee intake, however, is less prone to misreporting due to its long-term habitual nature. No data on mid-life coffee consumption was obtained, therefore potential deleterious or beneficial effects of coffee intake at midlife were not assessed. Lastly, the study was unable to differentiate between caffeinated and decaffeinated coffee, nor the benefits or consequences of preparation methods (brewing method, added milk and/or sugar etc.).
Conclusion

Increased coffee consumption potentially reduces cognitive decline by slowing cerebral Aβ-amyloid accumulation, attenuating the associated neurotoxicity from Aβ-amyloid-mediated oxidative stress and inflammatory processes. It is yet to be determined precisely which constituents of coffee are behind its positive effects on brain health and further research is required evaluating whether coffee intake could one day be recommended as a lifestyle factor aimed at delaying the onset of AD.
References


