The Effects of Caffeine on the Reduction of Amyloid-Beta Aggregation in Late-Onset Alzheimer's Disease

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TOHS AP Research STEM

Abstract

The effects of caffeine on concentrations of amyloid-beta proteins in late-onset Alzheimer's disease were investigated. Data was collected through a systematic literature review. Results of a statistical analysis produced a negative correlation between the concentrations of caffeine and amyloid-beta, suggesting that caffeine may play a role in reducing plaques in patients with late-onset Alzheimer's disease.

Introduction

Alzheimer's disease is a neurodegenerative disease affecting six million Americans today. It is a leading cause of death and has no apparent cure. The disease is characterized by neuron death arising from amyloid-beta protein plaques, preventing nerve communication and signaling in the brain. Caffeine, $C_8H_{10}N_4O_2$, is an extracellular particle that expresses a signaling pathway through molecular antagonism. It has been shown to reduce amounts of harmful proteins in the brain, which may apply to Alzheimer's disease because its plaques are made of toxic amyloid-beta 1-42 and 1-40 proteins.

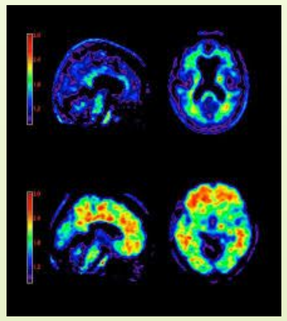


Fig. 1 The brain before and after Alzheimer's disease. Increased color indicates increased amyloid-beta.

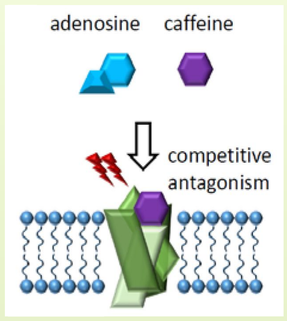


Fig. 2 Caffeine acting as an antagonist of adenosine by binding with adenosine receptors.

Purpose

The purpose of this study is to investigate the relationship between caffeine concentration and amyloid-beta 1-42 and 1-40 in the brain. Since these proteins make up Alzheimer's disease plaques, study results may assess if caffeine has a role in Alzheimer's development.

Research Question & Hypotheses

Is there a relationship between caffeine concentration in the brain and amyloid-beta protein levels seen in the late-onset Alzheimer's disease development?

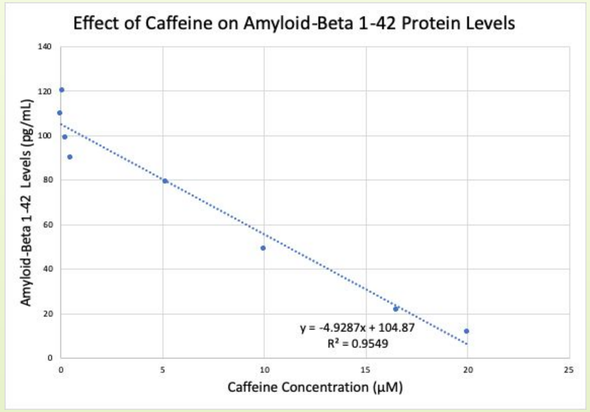
Null hypothesis: There is no relationship between neural caffeine concentration and amyloid-beta protein levels.

Alternative hypothesis: There is a statistically significant relationship between neural caffeine concentration and amyloid-beta protein levels.

Methodology

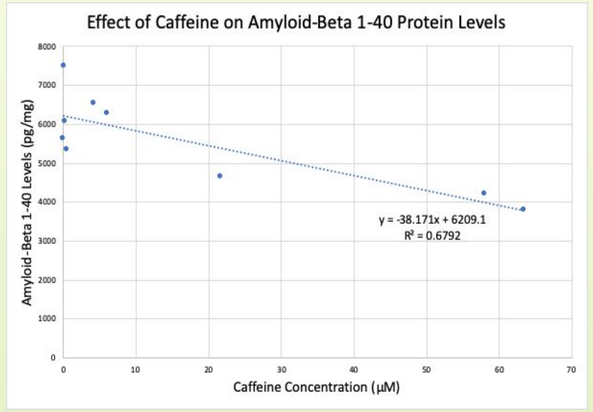
A systematic literature review was conducted for this study. The electronic bibliographic databases EBSCOhost, Google Scholar, PubMed, ResearchGate, Cochrane Library, JSTOR, and AAAS were used to collect literature. Quantitative data testing the effects of caffeine on the presence of amyloid-beta 1-40 and 1-42 proteins was compiled. The results were then studied, recorded, and analyzed with statistical testing.

Results



p = 0.0014

Fig. 3 Graph depicting amyloid-beta 1-42 levels as caffeine concentration increases as shown through eight observances. The best fit line and R-squared value are given.



p = 3.3E-07

Fig. 4 Graph depicting amyloid-beta 1-40 levels as caffeine concentration increases as shown through eight observances. The best fit line and R-squared value are given.

Discussion

In Figure 3, the linear regression produced a trendline with a 95.49% explanatory fit. The R-squared value, being close to 1.00, indicates a precise model, giving credibility to the shown correlation. A one-tailed t-test calculated a p-value of 0.0014, suggesting statistical significance. In Figure 4, linear regression produced a trendline with a 67.92% fit. This smaller value was due to scattered measurements from concentrations 0.0 to 6.0 μM, which were farthest from the line of best fit. A p-value of $3.3*10^{-7}$ was calculated using the t-test, indicating significance. The relationships in both sets of data are therefore statistically significant and support the alternative hypothesis.

Conclusion

Based on the above results, a negative correlation between amyloid-beta and caffeine is indicated and the alternative hypothesis is accepted.

Further Work

It would be beneficial to study the mechanisms of caffeine antagonism in amyloid-beta reduction, as well as the effects of caffeine on amyloid-beta in the long term to further apply results to Alzheimer's disease.

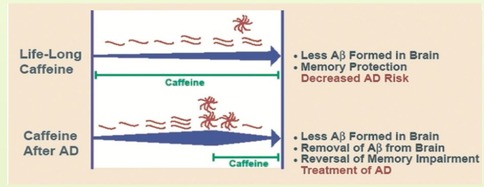


Fig. 5 Potential long-term effects of caffeine on Alzheimer's disease.

Research in novel drug therapies involving caffeine should also be considered to potentially lead to an effective Alzheimer's treatment.

Acknowledgments

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