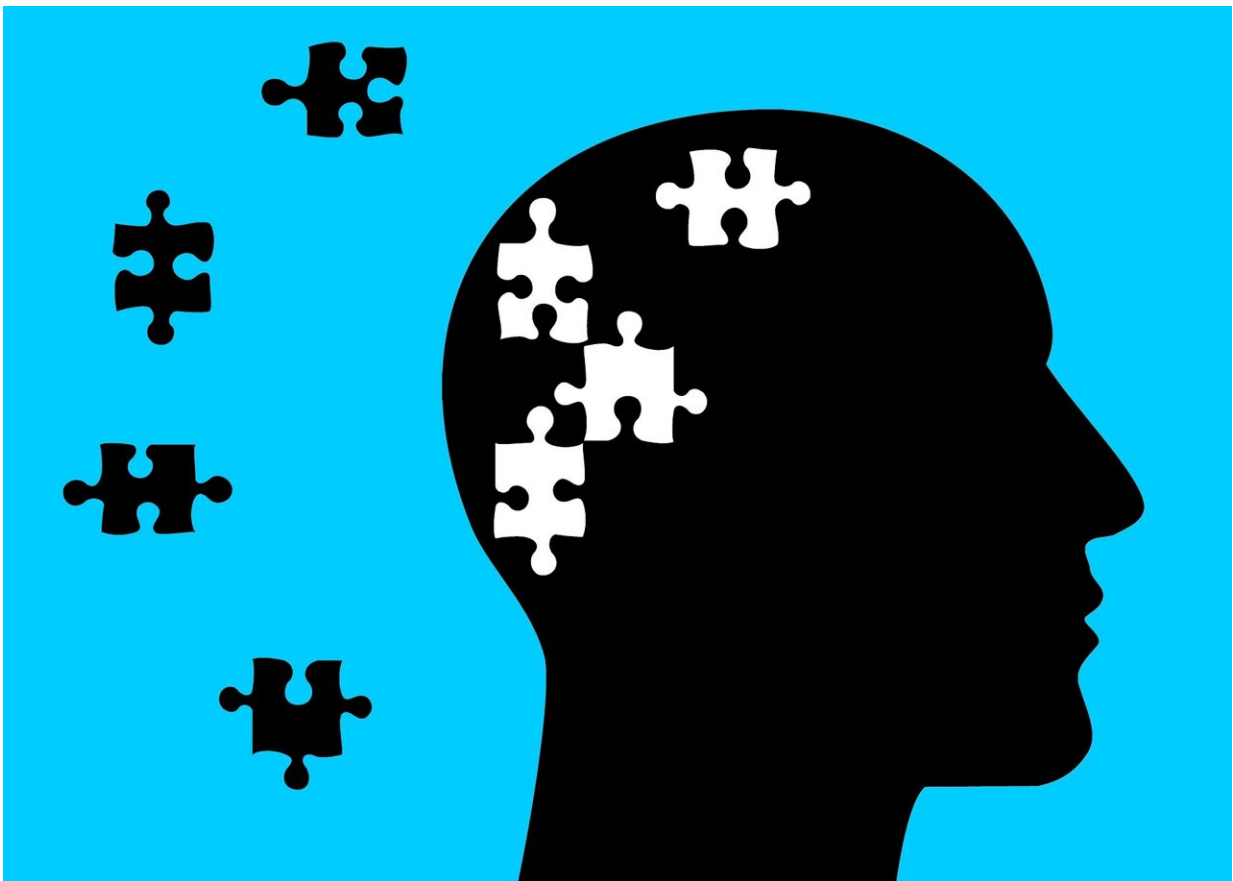


Clinical trial: Investigational anti-amyloid treatment started before Alzheimer's symptoms did not slow memory loss

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Preliminary results from a landmark clinical trial to prevent Alzheimer's

disease (AD) symptoms show that an investigational anti-amyloid drug, solanezumab, did not demonstrate a statistically significant slowing of cognitive decline associated with AD when initiated prior to the stage of clinical impairment.

The Anti-Amyloid Treatment in Asymptomatic Alzheimer's study ("A4 Study") is coordinated by the Alzheimer's Therapeutic Research Institute at the Keck School of Medicine of USC and is an affiliated project of the Alzheimer's Clinical Trials Consortium. The A4 Study was led by co-principal investigator Reisa Sperling, MD, Director of the Center for Alzheimer Research and Treatment at Brigham and Women's Hospital, a founding member of the Mass General Brigham healthcare system.

"Unfortunately, the results from our study did not show evidence that treatment with solanezumab slowed cognitive or functional decline at the preclinical stage of AD," said Sperling. "We are very disappointed for our participants and their families, as well as the hundreds of people who worked on this study for almost a decade, but we will learn a great deal from this work that will inform ongoing and future trials."

No statistically significant difference was observed between solanezumab and [placebo](#) groups on the primary outcome measure, the Preclinical Alzheimer Cognitive Composite (PACC) (mean change (95% CI): placebo -1.4 (-1.76, -1.04); solanezumab -1.69 (-2.13, -1.26); p-value=0.26). Secondary outcome results were consistent with the primary outcome, with all clinical outcomes numerically favoring placebo compared with solanezumab.

Longitudinal amyloid PET imaging demonstrated that amyloid continued to accumulate over time in both placebo (65.9 Centiloid baseline, 17.5 Centiloid increase) and solanezumab (66.2 Centiloid baseline, 12.1 Centiloid increase) groups. Higher baseline amyloid levels were strongly associated with a greater risk of progression to symptomatic Alzheimer's

disease (p-value

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