Elucidating the Impact of Lactobacillus casei ATCC 393 on Amyloid Plaque Generation in Alzheimer's Model Caenorhabditis elegans GRU102

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Alzheimer's disease, a progressive neurodegenerative disorder marked by the accumulation of beta-amyloid plaques in the brain, has become a global issue; One without a cure. Recent research highlights a possible link between gut microbiota and Alzheimer's development via the gut-brain axis. Common intestinal microorganisms, notably Lactobacillus Casei ATCC 393, generate compounds that influence brain function. These substances could hold neuroprotective attributes and potentially modulate processes implicated in Alzheimer's, including cognitive decline, neuroinflammation, and synaptic irregularities. It is imperative to investigate the role of Lactobacillus treatment in prospective therapeutic applications that address Alzheimer's pathogenesis. The study investigates the multifarious interactions of Lactobacillus Casei ATCC 393 within the Alzheimer's disease model that will yield an observable decrease in amyloid plaque agglomeration, while simultaneously improving cognitive function. To explore this, genetically modified Caenorhabditis elegans strains exhibiting Alzheimer's traits were age-matched and fed L. casei over a 38 hour time-period. Protein and tissue samples were collected from these organisms and utilized for an ELISA and a Thioflavin stain. Over the aging process these strains exhibited an escalating presence of amyloid plaques. Remarkably, the introduction of Lactobacillus casei ATCC 393 appeared to counteract this trend, suggestive of its potential to mitigate amyloid plaque accrual. These results provide strong support for the hypothesis, and furnish compelling insights into the plausible role of probiotics, such as L. casei ATCC 393, in ameliorating Alzheimer's-related phenomena, bridging the gap between gut microbiota and neurodegenerative disease.

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