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Abnormal Savda Munzi improves memory function in a APP/SP1 model of Alzheimer's disease by improvement of histology, ultrastructure and cholinergic system

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Alzheimer's disease (AD) is a debilitating neurodegenerative disorder characterized by increased b-amyloid (Ab) deposition and neuronal dysfunction leading to impaired learning and recall. Abnormal Savda Munziq (ASMq), a formulation has been used traditionally in Uighur medicine system of medicine as a memory enhancer and anti-stressagent. The present study was carried out to evaluate the ameliorating memory impairment in the transgenic mice and to explore the underlying molecular mechanism. Mice brain sections were detected by HE and transmission electron microscopy. Acetylcholine (Ach) levels, acetylcholineesterase (AchE) activity, Choline acetyltransferase (ChAT) activity were assessed in the hippocampus. ASMq treatment for 3 month decreased escape latency and swimming distance of mice from the third day in maze tests, and increased percent time in the target quadrant, along with increased Ach conceteration and improved histology and ultrastructure. The present study suggests that ASMq confers a therapeutic potential to ameliorate AD-like pathology in the brain of AD mice though both regulate histology, ultrastructure and cholinergic system.

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Microglia metabolizes β-amyloid 42 protein and produces a truncated neurotoxic fragment

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Microglia display a dual role in Alzheimer's disease (AD). At early stages of the pathology, microglia is beneficialby phagocytosing amyloid-β42 (Aβ42) and, at later stages, become detrimental, if the inflammatory response is not down-regulated. We provide evidence that microglia promotes the formation of a metabolized, toxic form of Aβ42 lacking its C-terminal region, as a result of the enzymatic activity of MMP-9. This N-terminal, truncated Aβ form is highly neurotoxic and more efficient than Aβ42 in inducing endogenous Aβproduction. Furthermore, it is able to promote aggregate formation in the brain of injected wild type mice, where Aβ42 has no effect. By the identification of this novel N-terminal, truncated Aβ form able to initiate amyloid aggregation *in vivo* even in the absence of mutations known to be associated with AD, our data provide the first evidence for the contribution of microglia to the development of the sporadic form of AD.

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