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Overexpression of Sirtuin3
improves survival and ameliorates
the pathological changes in a
mouse model of Alzheimer's
disease. Alzheimer's disease (AD)
is the most common
neurodegenerative disorder.
Various therapeutic strategies have
been developed to treat AD and
among them, the modulation of the
acetyl cholinesterase (AChE) and

BACE1 is considered the primary approach. However, several side effects such as off-target effects and the enormous efforts of development makes the development of novel drugs practically, time consuming and expensive. Recently, we have shown that sirtuin3 (SIRT3), a NAD-dependent protein deacetylase, which is also known as the anti-aging protein, improves the cognition of mice infected with *Listeria monocytogenes* (LM) and attenuates the LM-induced neuroinflammation. Based on these findings, we hypothesized that SIRT3 overexpression may improve the cognitive impairment in AD animal models, and further, we examined its neuroprotective properties in the Tg2576 mouse

model of AD. The results of behavioral assessments have shown that SIRT3 overexpression in Tg2576 mice improves their memory impairment. Moreover, the overexpression of SIRT3 decreases A β oligomerization, and decreases the phosphorylation of tau and β -site amyloid precursor protein-cleaving enzyme 1 (BACE1) levels and ameliorates pathological changes in the Tg2576 mouse model of AD.[Histopathological study of acne vulgaris]. To study the histological features of acne vulgaris and the potential etiology of acne. A histological study was conducted on tissues of the perilesional skin of patients with mild to moderate acne vulgaris by hematoxylin-eosin stain and immunohistochemistry stain for

eosinophil major basic protein. Acne vulgaris is an c6a93da74d

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