MEF2C

Myocyte enhancer factor 2C (MEF2C) is highly expressed in the nervous system, and regulates neuro-development, synaptic plasticity, and inflammation. However, its mechanism in Alzheimer's disease (AD) is underestimated. In a study, the role and mechanism of MEF2C were investigated in the brain tissue specimens from patients with AD, APPswe/PSEN1dE9 double transgenic (APP/PS1_DT) mice, and SH-SY5Y cells treated with amyloid beta peptide (Aβ). The results indicated that the expression of MEF2C is significantly reduced, and the expression of MEF2C/Aβ in different parts of brain is negatively correlated in patients with AD. Knockdown of MEF2C promotes cell apoptosis and the level of β-amyloid precursor protein cleaving enzyme 1 (BACE) but reduces BACE2 expression. In addition, knockdown of enhances the generation and aggregation of Aβ in the cortex of APP/PS1_DT mice, reduces the expression of synaptic proteins, exacerbates the ability of learning and memory of APP/PS1_DT mice, damages the structure of mitochondria, increases the oxidative stress (OS) level, and inhibits the expression levels of members of the Nrf2-ARE signal pathway. In summary, inhibition of MEF2C exacerbates the toxic effect of Aβ and , damages synaptic plasticity, reduces the ability of learning and memory of APP/PS1 mice, and increases the level of OS via the Nrf2-ARE signal pathway [1].

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