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Investigation of the effect of melatonin on the aggregation of proteins

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Abstract

Alzheimer's disease is one of the most common degenerative diseases of the nervous system that disrupts the cognitive and behavioral abilities of its sufferers. Alzheimer's has pathological symptoms including extracellular senile plaques mainly consist of aggregated amyloid beta fibrils, intracellular neurofibrillary tangles consisting of hyperphosphorylated tau protein, oxidative stress, and neuroinflammation. In most people with Alzheimer's, circadian rhythm disorders that are the basis of sleep-wake cycle disorders have also been seen, and there is a two-way relationship between Alzheimer's and sleep disorders. Melatonin is a hormone that regulates the sleep-wake cycle, circadian rhythm, and sleep homeostasis, it also acts as a scavenger of free radicals and an antioxidant and causes the differentiation and proliferation of nerve cells. The level of melatonin gradually decreases with age, and elderly people secrete the least amount of melatonin, which is considered as an important factor in the development of Alzheimer's. Since melatonin has anti-aggregation properties, in this study, using fluorescence and absorption spectrometry methods, fluorescence imaging, and atomic force microscopy, investigating possible interactions with the molecular docking method, and its comparative effect on the aggregation of proteins was investigated. Also, the antioxidant effects of melatonin on the treated SH-SY5Y cell line with protein aggregated were studied using cell viability, reactive oxygen species, and mitochondrial membrane potential. The results showed that melatonin, with its anti-amyloid properties, can reduce the formation of protein aggregates, and considering the docking findings, it seems that there is a molecular interaction between melatonin and the proteins. Also, the results of this research showed that due to its antioxidant properties, melatonin was able to increase survival, reduce reactive oxygen species, and reduce mitochondrial membrane damage in cells treated with aggregates.

Key words: Alzheimer's disease, protein aggregation, melatonin, sleep





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