

Evaluation of the effectiveness of some benzoxazole derivatives in the experimental Alzheimer's models in vitro

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Alzheimer's disease (AD) is a common primary health problem, especially in the aging population. AD is a neurodegenerative disease characterized by loss of neurons and synapses, with extracellular plaques containing A β and intracellular neurofibrillary tangles containing tau. In this study, we were evaluated the preventative effect of the A β aggregation in the SH-SY5Y cells of the three different benzoxazole derivative compounds, which were called compounds 1, 2, and 3. After 24 hours of 40 μ M A β incubation, SH-SY5Y cells were treated for 24 and 48 hours with the three different benzoxazole derivative compounds separately and the proliferation of SHSY-5Y cells were assessed using the MTT assay. We observed significant neurite inhibition with moderate damage by the NST at IC₅₀ dilutions of the compounds. Then AD's disease related A β plaque analyzed for the by microscopic and FOXA2 and PEN2 markers in vitro by ELISA methods. At the end of the 24th and 48th hours, compound 2 and 3 treated cells were shown reduced A β plaque formation, while compound 1 treated cells were shown the same A β plaque formation manner according to the non-treated A β control group. However, compound 3 treated cells showed a higher reduction rate on the A β plaque formation in 40 μ M A β treated cells. Also, FOXA2 cytokine level was found to significantly decrease in the compound 2 treated cells. In contrast, FOXA2 cytokine level significantly increased in the compounds 1 and 3 treated cells according to the non-treated A β control group. On the other hand, the PEN2 cytokine level was significantly decreased in the compound 1, 2, and 3 treated cells. Additionally, neurite extension assays revealed that the compounds 3 treated cells were shown higher neurite extension rates. Thus, among the heterocyclic compounds we used, the compound 3 significantly prolongs the neuritis and may have a therapeutic effect while reducing beta-amyloid accumulation in AD's patients.