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Modifying the Effects of Aβ40 and Aβ42 Proteins in Patients with Alzheimer’s Disease

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Amyloid Oligomers

Neuronal Cultures

and is the 6th leading cause of death in the United States. Alzheimer’s Disease affects 1 in 3 seniors, Figure 1. Alzheimer’s Disease Facts

United States.

Synapse Counting

should act as a therapy for neurodegeneration and age-dependent learning defects. This modification will not interfere with neuron signaling. This modification should act as a therapy for patients.

Specific Aim: My specific aim is to modify Amyloid-Beta-40 (Aβ40) and Amyloid-Beta 42 (Aβ42). This is so the proteins will not interfere with neuronal signaling. This modification should act as a therapy for patients.

Proposed Methodology

- Collect rat embryos and extract their Hippocampus and Cortical tissue. Add 2.5% Trypsin to dissociate the cells. Place cells in 384-poly D-Lysine coated plates with Neurobasal Media, Glutamax, and antibiotics.
- For Synthetic Abeta 40/42 oligomer preparation, add oligomers an hour prior to the compounds. For 15 minutes, fix the cells with 0.5% of Triton X-1003, 75% formaldehyde and block with 5% goat serum. Incubate with the primary antibodies for Synaptophysin and Abeta, MAP-2.
- Before adding 6 mM of synthetic Abeta 40/42 oligomers, treat cultures with compounds. Then, incubate for 24 hours. Use the ThermoFisher/Cellomics Neuronal Profiling bio application to count the Synaptophysin-immunopositive puncta.
- The well averages will be looked at with the KS distance test. The Abeta 40/42 oligomers will be observed with the Western Blot Analysis. The treatment differences will be looked at with One-way ANOVA.

Experimental Results

Figure 3. Hippocampal/cortical cultures immunofluorescent labeling. A. Labeled MAP2 neurons. B. Labeled DAPI nuclei. C. Labeled GFAP glia and Nuclei. D. This image displays the merging parts of all three images. The percentage of neurons was 26.0 ± 1.1% in the cultures. This was based on the untreated control wells. (Izzo 2014) The primary rat neurons can replicate the electrophysiological state-dependent signaling.

Figure 4. Plaques and Tangles

These illustrations display the process of how plaques and tangles function in the brain. A. The top picture shows where plaques are located on the neuron. The bottom picture shows how tau are attached to the microtubules. B. This illustration shows the atrophic affects of the brain from plaques and tangles.

Discussion/ Conclusions

Potential Pitfalls:

- Creating a therapy could cause issues with patients.
- There could possibly be failure to provide therapeutic benefits for patients with Alzheimer’s Disease.
- There could also be a failure in therapy if the patient has undergone too much neuronal loss.

Potential Conclusions:

- The symptoms of neurodegeneration could decrease with modifications done to the Amyloid Beta-40 and 42 proteins.
- This could serve as a therapy for AD patients and save people from losing their memory entirely.
- A cure still hasn’t been found for Alzheimer’s, so it’s important to continue researching this insufferable disease.

Literature Cited

