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β-Amyloid (1-42) and Its Role in Developing an Animal Model of Alzheimer's Disease

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ß-AMYLOID (1-42) AND ITS ROLE IN DEVELOPING AN ANIMAL MODEL OF ALZHEIMER'S DISEASE

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Alzheimer's disease is a neurodegenerative disorder characterized by neurofibrillary tangles and neuritic plaques. The major component of neuritic plaques is ß-amyloid. In humans, ß-amyloid is a chain of amino acids varying in length from 39 to 43. Current studies have shown that ß-amyloid (1-40) is toxic to cells *in vitro* , with the 25-35 chain of amino acids supposedly being responsible for the cell death. However, the ability of the protein to cause cell death *in vivo* is still being questioned. One problem is that the (1-40) form has been found to aggregate very rapidly in most solvents, which could prevent a successful injection of ßA into a rat. Recently, the (1-42) form has been purified and synthesized. As opposed to the (1-40) form, the new 42 amino acid sequence has a slower rate of sedimentation, which increases the likelihood of an effective injection. In order to determine the effect of ßA (1-42) on spatial tasks, 0.5 microliters of the peptide were injected bilaterally into the dorsal hippocampus of male Long Evans rats. Control groups were also utilized. The first control group consisted of injections of a scrambled version of the peptide, the second group being the vehicle (DMSO) alone. The animals, which had been pretrained on a radial arm maze, were allowed to recuperate from surgery and were then tested on the maze again. The maze consisted of eight arms, five of which were always baited for a particular animal. Session latency, latency to first choice, number of correct choices, number of reference memory errors, number of correct and incorrect errors, and the total number of choices were recorded for each animal. Items that were then calculated included the percent of correct choices, the average choice latency, and the total number of errors. Testing continued for two weeks, after which the reinforcers from three of the arms were removed and shifted them to the three arms which had previously been unbaited. Three days later, all reinforcers were removed except for the two arms which had been baited for both of the previous configurations. These two reconfiguration procedures were done in an attempt to differentiate between animals using procedural memory and those relying on declarative memory, which is controlled by the hippocampus. The data was analyzed using ANOVA. The rats were perfused after testing, and the histologies are currently being completed. There was no significant difference between the three groups. These results suggest that injections of ßA do not affect the retention of previously learned spatial tasks in the rat. Further studies will assess the affects of bilateral injections of ßA (1-42) into the hippocampus on the acquisition of a spatial learning task.