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AN ANIMALS MODEL OF ALZHEIMER'S DISEASE III: CHARACTERIZATION OF NEURONAL DEGENERATION USING THE TECHNIQUE OF IMMUNOCYTOCHEMISTRY FOR GLIAL FIBRILLARY ACIDIC PROTEIN

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Alzheimers disease (AD) currently affects an estimated 4 million Americans. Manifested initially by mild forgetfulness, this devastating disease eventually erodes all cognitive abilities. Neuropathologically AD is characterized by neuritic plaques (NP) and intracellular neurofibrillary tangles (NFT). Although the etiology of AD is unknown, NP deposition has been suggested to play a fundamental role. Recently, the major component of NP has been identified as a B-amyloid protein that exists in an insoluble state. This pathology is invariably accompanied by the proliferation of adjacent gial cells in response to neuronal degeneration. In particular, postmortem studies of AD patients show a marked increase in the level of glial fibrillary acidic protein (GFAP) due to astrogliosis. Moreover, recent development of immunocytochemical techniques which stain specific proteins, have shown strong correlation between the degree of dementia in AD patients with the degree of intensity of GFAP staining. These results strongly suggests that immunocytochemisty for GFAP is a reliable tool in measuring neurodegeneration in AD patients

In a study that is being conducted concurrently (see David Kang's abstract), intrahippocampal injections of Beta-Amyloid (25-35) or Ibotenic Acid have been used to induce a potential animal model of AD. In this study GFAP immunocytochemistry will be employed to assess the degree of neuronal degeneration in these animals. The result of this assessment will be presented at the conference.