Alzheimer Disease Pathobiology and Mechanisms of Neurodegeneration

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Canada

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Alzheimer Disease Pathobiology and Mechanisms of Neurodegeneration

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Research Abstract

Alzheimer's disease (AD) is a common, progressive and ultimately lethal disease affecting ~5% of adults over 65 years of age and ~30% of adults over the age of 85 years in most Western societies. Both the prevalence and the annual expenditures on AD are expected to increase as the proportion of the population at risk increases over the next 20 years. The currently available treatments are of limited effectiveness as they target the symptoms of the disease but not the root cause. The major features of AD are accumulations of abnormal proteins called amyloid plaques and neurofibrillary tangles within the brain. Amyloid plaques appear as densely packed thread-like fibers that form in the vicinity of nerve cells and blood vessels. Evidence suggests

that amyloid is highly toxic and is the primary cause of nerve cell death. Loss of these cells is the underlying event in AD and results in the clinical features of cognitive dysfunction and memory impairments. We have identified the presenilin family of proteins which are the major cause of familial or inherited Alzheimer's disease. Presenilins are unique enzymes and are part of a larger complex (termed gamma-secretase) which directly produces the toxic amyloid in the brain. Presenilins are intimately involved in the disease process as shown by the fact that mutations or changes in their structure accelerates amyloid formation and results in early onset AD (25-40 years of age). However, the exact mechanism by which this is achieved remains unclear. Our research will investigate the molecular organization of the presenilins and their functional partners. This will yield valuable information on how they function and, with this research is to understand the cellular processes which cause AD and to use that information for the development of safe and effective treatments.

Further information available at:

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