# AGE-RELATED VASCULAR DEGENERATION AND MEMORY DECLINE

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**Background:** Concepts of the pathogenesis of late-life dementia have significantly changed over the last century. Although Alzheimer described a primary role of atherosclerosis in late-life dementia, the concept of amyloid toxicity-induced neuronal degeneration has dominated the field of aging research for decades. While clinical observations suggest the contributory role of vascular dysfunction in late-life dementia, aging research appeared to underestimate the importance of the vascular components in brain degeneration.

**Method:** Two groups of rats (male fisher/NB), young (6m, n=10) and aged (35m, n=10) were used. Morris Water Maze tests were carried in both age groups. There were 4 trials in each 4 days of acquisition tests and the starting positions were randomly changed between the training days, but remained the same in each trial. A probe trial (30s) was carried out immediately after the last acquisition test. The brain tissues were collected for histological and Immunohistochemical staining.

**Results:** The aged rats had significantly impaired memory compared to the young rats. The aged rats also showed the degenerative changes and the loss of the capillaries in the hippocampus compared to the young controls. The memory decline was also related to the loss of GFAP and Iso-lectin B4 positive glial cells and white matter density. There was no neuronal degeneration in the hippocampus of aged rats.

**Conclusion:** In accordance of clinical observations, our data suggest that late-life dementia might be a vascular disorder with a consequence of neurodegeneration.