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Science

Dementia is generally considered to be one of the most burdensome and disabling conditions of later life. Besides the severe impact it has on a patient's life, it also has profound socio-economical consequences for the caregivers and society in general. In 2005, the number of patients with dementia was estimated at 24,3 million worldwide with an annual incidence rate of 4-6 million. In the coming decades these numbers are expected to increase drastically due to the increase in life expectancy and the ageing of the population. The most common cause of dementia is Alzheimer's disease (AD), which causes over 50% of all cases. AD is a neurodegenerative disease with two characteristic histopathological lesions: amyloid plaques and neurofibrillary tangles of hyperphosphorylated tau. The symptoms comprise a wide variety of cognitive, behavioural and psychological disturbances. The exact pathophysiological processes underlying the lesions and symptoms are, however, still unknown. A better knowledge of these processes could contribute to the development of effective treatments and diagnostic tools. At the moment all available treatments are still only symptomatic and a definitive diagnosis can only be made after autopsy. Additional research in this area is therefore crucial. During these last decades compelling genetic and biochemical evidence has been brought to light, suggesting that the amyloïd-beta protein (Aβ) plays a central role in the AD pathology, in particular the smaller, soluble aggregates. In addition, recent research has shown that the degenerating neurons in AD show signs of DNA synthesis and express proteins related to the cell cycle. These findings led to the hypothesis that terminally differentiated neurons might attempt to reactivate the cell cycle in AD. Both the soluble amyloid beta as well as the cell cycle events (CCE) could represent some of the earliest processes of the AD pathology and could prove interesting for the

development of treatments and “early stage” detection methods. Therefore, we will use western blotting, fluorescence microscopy and behavioural and cognitive testing to further study these elements, their relationship to each other and their relationship to Alzheimer’s disease. Given the current limitations of human brain research, we will use a validated animal model for alzheimer’s disease, the transgenic APP23-mouse model, for this project.