

Investigating possible alterations in the NG2 cell population brain tissue from Alzheimer's disease patients and healthy elders

Malin Wennström et al.

Clinical Memory Research Unit, Dept. of Clinical Sciences, Lund University, Malmö, Sweden.

Research question and background

Neuron Glia 2 (NG2) cells constitutes the major group of cells undergoing mitosis in the adult rodent brain representing 5%–8% of all cells in the nervous system. The cells are recognized by their stellate morphology and expression of the chondroitin sulphate proteoglycan termed Neuron-Glia 2 (NG2). The function of NG2-cells is largely unknown, but studies on rodents have shown that they communicate with nearby cells, make synaptic connections with neurons and play a role in the guidance of axonal outgrowth. In response to CNS damage NG2-cells become activated, acquire an amoeba-like phenotype and secrete pro and anti-inflammatory agents. To date there are few studies describing the role of NG2 cells in the healthy and diseased human brain. In the current study therefore we aim to investigate possible alterations in the NG2 cell population brain tissue from AD patients and healthy elders.

Methods and tissues used

Tissue from entorhinal cortex and hippocampus were collected from patients diagnosed with AD (n=5) and healthy elder controls (n=7). Tissue sections were immunohistochemically stained for NG2 as well as A β 1-42 and hyperphosphorylated tau. NG2 cell Immunoreactivity, morphology and distribution in relation to amyloid plaque and tangles were thereafter analyzed using a light microscope.

Results and conclusion

NG2 cells controls without A β deposits displayed multiple thin, branched processes, whereas most of the analyzed NG2 cells in individuals with A β plaques displayed fewer, less branched and shorter processes and swollen cell bodies. Moreover, the NG2 staining in AD patients with high plaque load was barely visible, indicating low NG2 expression in these patients. The NG2 cells were evenly distributed and were not found to cluster around A β plaques. Our results demonstrate alterations in the NG2 cell population in relation to AD pathology, which highlights the NG2 cell population as a new attractive research target in the search for cellular mechanisms associated with AD pathogenesis.