Neuroinflammation: Implications for the Pathogenesis and Molecular Diagnosis of Alzheimer's Disease

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During the past few years, an increasing set of evidence has supported the major role of deregulation of the interaction patterns between glial cells and neurons in the pathway toward neuronal degeneration. Neurons and glial cells, together with brain vessels, constitute an integrated system for brain function. Inflammation is a process related with the onset of several neurodegenerative disorders, including Alzheimer's disease (AD). Several hypotheses have been postulated to explain the pathogenesis of AD, but none provides insight into the early events that trigger metabolic and cellular alterations in neuronal degeneration. The amyloid hypothesis was sustained on the basis that A?-peptide deposition into senile plaques is responsible for neurodegeneration. However, recent findings point to A? oligomers as responsible for synaptic impairment in neuronal degeneration. Amyloid is only one among many other major factors affecting the quality of neuronal cells. Another explanation derive