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ACTIVATED CASPASE-3 EXPRESSION IN GLIAL CELLS AFTER AN EXCITOTOXIC LESION IN THE OLD RAT BRAIN.

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Little is known about the mechanisms triggering glial activation and those downregulating glial reactivity after the inflammatory process. These mechanisms may be altered in the aged brain and may underlie the increased glial responsiveness that enhances the aged brain's susceptibility to injury and neurodegenerative diseases. Recent in vitro studies suggest that cell death may be the underlying mechanism for the auto-regulation of the glial response. Caspase-3 is one of the key executioners of apoptosis, being responsible for the proteolytic cleavage of many key proteins. In this study we evaluate the expression of caspase-3 after an experimental excitotoxic lesion (injection of N-methyl-D-aspartate) in the striatum of 2 year old rats. Animals were sacrificed at different times between 1 and 14 days after the lesion and frozen coronal sections were processed immunohistochemically for the demonstration of activated caspase-3. In addition double labelling with GFAP (astroglial marker), tomato lectin (microglial marker) and neuronal nuclear antigen (Neu-N) was performed for the identification of caspase-3 positive cells. Unlike control animals, where only scattered caspase-3 immunolabeled cells are observed, after the striatal excitotoxic injury, activated caspase-3 immunoreactivity is present in the ipsilateral striatum at all time points studied, correlating with the areas of reactive gliosis. Maximal caspase-3 expression is observed at 3 days post lesion, when most positive cells are identified as reactive astrocytes. Our results indicate that apoptotic mechanisms may be involved in controlling the extent of reactive astrogliosis following an excitotoxic injury to the aged brain. Supported by Fundació marató TV3, 'la Caixa'and DGES PB98-0892.