PROLIFERATIVE RESPONSE OF GLIAL CELLS IN AREAS OF PRIMARY AND SECONDARY DEGENERATION FOLLOWING A NEOCORTICAL ASPIRATION LESION IN THE ADULT RAT BRAIN

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The focus of the present work was to study the proliferative response of glial cells associated with the microglial and astroglial reactivity both in areas of primary degeneration (neocortex) and in areas undergoing secondary degeneration (thalamus) after an aspiration lesion affecting the hindlimb area of the sensorimotor cortex in the adult rat. After survival times ranging from 12 hours to 30 days, animals were sacrified by intracardiac perfusion, the brains were removed and embedded in paraffin, and coronal sections (10 μm thick) were obtained and processed for the immunocytochemical demonstration of the proliferating cell nuclear antigen (PCNA). The proliferative cells were identified by double labeling techniques combining immunodetection of PCNA with selective glial markers: tomato lectin histochemistry for the demonstration of microglia and immunocytochemical detection of glial fibrillary acidic protein for the demonstration of astrocytes.

Glial cells proliferate both in cortical areas of primary degeneration as well as in thalamic nuclei undergoing secondary degeneration: laterodorsal-ventrolateral nucleus (LDVL), laterodorsal-dorsomedial nucleus (LDDM), reticular nuclei (Rt), ventral-posterolateral nucleus (VPL), posterior (Po) nucleus and antero-ventral nucleus (AV). However, the temporal pattern and the intensity of the proliferative response differ in function of area and the glial cell type considered. The proliferative response of glial cells is earlier and more intense in cortical areas adjacent to the lesion site than in the secondarily affected thalamic nuclei. In addition, differences in the pattern and the degree of the glial proliferative response were observed when the different affected thalamic nuclei were compared.

The proliferative response of microglial cells precedes and is more intense than that of astroglial cells both in primary and secondary degeneration areas. Proliferation of microglia is already evidenciable at 12 hours postlesion (PL) in the lesioned cortex and at 3 days PL in the affected thalamic nuclei. In contrast, the proliferation of astroglia is retarded and it is not appreciable until 2 days PL in the cortex and 7 days PL in the thalamus.

These differences in the temporal pattern of glial proliferation correlate perfectly with the pattern of tissular affectation and subsequent glial reactivity observed in both primary and secondary degeneration areas. This work was supported by DGICYT grant Nº PB95-0662 and Marató TV3.