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CORTICAL AND THALAMIC GLIAL RESPONSE IN THE EXCITOTOXICALLY LESIONED YOUNG BRAIN. L. Acarin*, B. González, A. J. Castro and B. Castellano. Unitat Histologia (Medicina), Univ. Autònoma Barcelona, Spain; and Dept. Cell Biology, Neurobiology and Anatomy, Loyola Univ. Chicago, U.S.A.

Experimental models using excitotoxic cortical lesions cause secondary changes of thalamic nuclei projecting to the damaged area, providing an useful tool for the comparison of glial reactivity between areas of direct lesion and secondary affected areas. The injection of the excitatory aminoacid analog NMDA into the right sensorimotor cortex of postnatal day nine rats caused secondary changes accompanied by glial reactivity in the thalamic ventrobasal complex (VB). After different survival times from 4 hours to 30 days, parallel cryostat sections were immunocytochemically processed for the demonstration of GFAP, vimentin, MHC I and II and serum proteins. Tomato lectin histochemistry was used to visualize microglia.

Results showed differences between the glial response in the cortex and the secondary glial response in VB complex of the thalamus. In the cortex, where serum proteins were detected from 4 hours to 7 days, microglial response was restricted to the degenerating area, was characterized by a rapid activation already observed at 4 hours post-lesion, before neuronal degeneration was evident, and peaked at day 3. Reactive microglial cells showed changes in morphology, increase in tomato lectin binding and MHC I expression. Additionally, few cells also expressed MHC II. Astroglial reactivity was more protracted, starting by day 1 post-lesion and increasing gradually before presenting its maximum at 7 days post-lesion. Hypetrophied reactive astrocytes showed increased GFAP expression and were vimentin positive.

Glial response in the VB complex of the thalamus occurred in the absence of apparent neuronal degeneration and serum proteins, and it was not as pronounced as in the excitotoxically lesioned cortex. Reactive microglial cells showed a bushy morphology, were intensely lectin positive and expressed MHC I, but not MHC II. The astroglial response remained longer than microglial reaction. Reactive astrocytes showed thick processes and increased GFAP expression, although no vimentin expression was seen. Funded by DGICYT PB95-0662, "la Caixa" fellowship program.