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Astroglial and microglial response in the thalamus after an excitotoxic neocortical lesion in the young brain.

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Cortical lesions cause secondary degeneration of thalamic nuclei projecting to the damaged area. Thus, this thalamic degeneration provides an experimental model for the comparison of glial reactivity between areas of direct lesion and secondary affected areas. Nine day old rats received an injection of NMDA into the right sensorimotor cortex, causing retrograde neuronal degeneration accompanied by glial reactivity in the ventrobasal complex (VB) of the thalamus. After different survival times ranging from 4 hours to 30 days, parallel cryostat sections were immunocytochemically processed for the demonstration of GFAP, vimentin and MHC I and II. Tomato lectin histochemistry was used for the demonstration of microglia.

Results showed differences between the glial response in the cortex and the glial response in VB complex of the thalamus, affected by secondary degeneration.

In the cortex, microglial response was restricted to the degenerating area and was characterized by a rapid onset. Microglial reaction was first seen at 10 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 5 and 7 days post-lesion. Hypetrophied reactive astrocytes showed increased GFAP expression and were vimentin positive.

Glial response in the VB complex of the thalamus was not as pronounced as in the excitotoxically lesioned cortex, although temporal delay was not observed. Reactive microglia showed a bushy morphology, were intensely lectin positive and expressed MHC I. Mild MHC II expression was only found at the onset of microglial activation. Astroglial response remained longer than microglial reaction. Reactive astrocytes showed thick processes and increased GFAP expression, although no vimentin expression was seen.

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