GLIAL EXPRESSION OF HEAT SHOCK PROTEIN 27 AND NFkB AFTER CORTICAL ASPIRATION LESION.

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A member of the small heat shock protein (sHSP) family, the 27KDa Heat shock protein (Hsp27), is induced in response to different insults (heat, oxidative stress, excitotoxicity, etc.). This protein acts as molecular chaperone, induces thermotolerance, maintains actin stability and protects against cytokine-induced cytotoxicity. In vitro studies showed that Hsp27 is able to inhibit NFkB nuclear translocation, a transcription factor involved in the activation of glial inflammatory response.

In this study, aspiration lesions of the sensorimotor cortex were performed in postnatal day nine rats. After survival times from 4 hours to 7 days, rats were perfused and brains were cut in a cryostat. Sections were processed by free floating immunohistochemistry for Hsp27 and NFkB detection.

Hsp27 was first observed at 1 day post lesion (PL), in astrocytes and microglial cells located in the penumbra-like area that surrounds the neuronal region in the borders of lesion. Double immunohistochemistry showed that Hsp27 immunoreactive glial cells had activated NFkB in the cytoplasm, but not in the nucleus. From 3 days PL, Hsp27 was progressively restricted to the borders of lesion where glial scar begins to form. In this location, the number of Hsp27 positive cells coexpressing nuclear NFkB showed a progressive increase.

Our results show that glial expression of Hsp27 may be involved in the regulation of NFkB translocation to the nucleus, controlling the deleterious effects of inflammatory response promoted by reactive glial cells in the penumbra-like area and the glial scar formation at the borders of lesion.

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