Expression of the type 1 and type 2 receptors for tumor necrosis factor after traumatic spinal cord injury in adult rats

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Abstract

Posttraumatic inflammation has been implicated in secondary tissue damage after spinal cord injury (SCI). Tumor necrosis factor-alpha (TNF-alpha) is a key inflammatory mediator that is increasingly expressed after SCI. The effect of TNF-alpha is mediated through its receptors TNFR1 (p55) and TNFR2 (p75). However, whether these two receptors are expressed after SCI has not been demonstrated. In the present study, the temporo-spatial expression of TNFR1 and TNFR2 was examined in rats that had received a 10 g impact injury dropped at a height of 12.5 mm using the New York University impact device. In sham operates, no detectable TNFR1 or TNFR2 immunoreactivity (IR) was observed. In contused spinal cord, TNFR1 protein expression and immunoreactivity (IR) were detected as early as 15 min postinjury, reached its peak at 8 h, and declined markedly after 1 and 3 days postinjury. The temporal pattern of TNFR2 expression was similar to that of TNFR1 but its expression peaked at 4 h postinjury. During peak expression, TNFR1- and TNFR2-IR were most intense at the site of injury and decreased gradually from the injury epicenter. TNFR1- and TNFR2-positive cells included neurons, astrocytes, and oligodendrocytes. Methylprednisolone (MP), a synthetic glucocorticoid, partially inhibited the injury-induced expression of TNFR1 and TNFR2, an effect which could be reversed by RU486, an antagonist of glucocorticoid receptors. We suggest that the expression of TNFR1 and TNFR2 after SCI may contribute to posttraumatic inflammatory responses of TNF-alpha.