FOOD RESTRICTION ALTERS INFLAMMATION AND APOPTOSIS DURING MULTIPLE CORTICAL INJURIES


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Introduction: The inflammation is the main cause of neuronal cell death after cortical injury. Numerous studies proved that food restriction (FR) can be beneficial and neuroprotective, in different pathological conditions.

Aims: To investigate whether FR lasting 3 months prior to cortical injury can modulate processes of inflammation and neuroapoptosis in the ipsilateral cortex, we examined TNF-α and caspase 3 expressions. We also followed two transcriptional pathways (p-GR and NFkB) and antiapoptotic genes involved.

Methods: Two groups of Wistar rats were used in the study: ad libitum (AL) and FR animals (50% of normal daily food intake 3 months prior to injury). Unilateral cortical multiple injuries to sensorimotor cortex were done. The tissue was collected at 2nd, 7th, 14th and 28th day post injury. The Fluoro-Jade B and Hoechst 33258 staining was used to show degenerating neurons.

Results: Western blot analysis showed the high level of TNF-α and caspase 3 expression only in AL group at 2nd day after injury. The expression was not detected in FR group at any time point. The Fluoro-Jade B and Hoechst staining confirmed neuronal cell death in 2nd day following injury but just in AL group. Upregulation of serum corticosterone and his active receptor p-GR was detected barely in FR group. Moreover, NFkB and downstream antiapoptotic genes were significantly upregulated in FR group, while downregulated in AL group.

Conclusions: Our results suggest that FR as pretreatment can abolish inflammation and apoptosis in the injured CNS tissue, perhaps through synchronized activation of GR and NFkB pathways.