TRANTHYRETIN RESPONDS TO STRESS IN RATS

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Introduction: Transthyretin (TTR) sequesters amyloid β (Aβ) preventing Aβ aggregation and fibril formation and deposition, thereby playing a protective role against Alzheimer’s disease (AD). Cortisol is a glucocorticoid hormone involved in metabolism, especially in the central nervous system in which it is related to stress. Previous studies showed that the induction of a stress response was used with success to prevent neurodegeneration.

Aim: In this context, we studied the effects of stress in TTR expression in rats submitted to overcrowding and in a cell line of rat choroid plexus (RCP) treated with hydrocortisone.

Methods: Animals (n=30) were submitted to a crowding stress experiment, by joining them in a cage (n=9/each treated group). RCP cells were incubated, in 5 time periods, with hydrocortisone (0, 10, 100, 1000 nM) and TTR expression was analyzed by Western blot.

Results: The incubation of RCP cells with hydrocortisone at 10, 100 or 1000 nM increased the expression levels of TTR after 12, 18 and 24 h of incubation. This up-regulation was more pronounced after 12 h using 100nM hydrocortisone. In animals, TTR expression was increased in rats subjected to stressful conditions (p < 0.001) and it was more pronounced in males than in females.

Conclusion: We concluded that stress stimulus increases TTR expression. This increase is important because it could interfere with the metabolism of TTR and its neuroprotective action against AD.