ABSTRACT

Dissertation deals with the influence of corticoliberin and corticosterone on behavior, memory and learning and the histomorphological changes in the hippocampus. The experiment was performed on rats – long- evans strain, line AV ČR, Krč (male, age ca three months, weight ca 350g).

The hormones used were administrated continuously; corticoliberin by the osmotic pump into the right lateral cerebral ventricle for four weeks, corticosterone by the subcutaneous pellet for three weeks. The animals were divided into three experimental groups and one control group. The group \([\text{↑CRH}]\) (n=6) osmotic pump-applied corticoliberin (1,5 µg per animal per day), it was made adrenalectomy with substitution by corticosterone subcutaneous pellet (1,4 mg per animal per day). The group \([\text{↑CRH↑CS}]\) (n=11) osmotic pump-applied corticoliberin (1,5 µg per animal per day). The third experimental group \([\text{↑CS}]\) (n=10) corticosterone was administered by subcutaneous pellet (9,52 mg per animal per day). The control group (n=10) was without any treatment. All groups were subjected to four various behavioral tests - test of active allothetic place avoidance (AAPA), step- through test, conditioned taste aversion (CTA), and test in Morris’ water maze (MWM).

In the \([\text{↑CRH}]\) group was found extension of habituation period caused by anxiety and prolongation of learning and a damage of long-term memory too. In the \([\text{↑CRH↑CS}]\) group primarily cognitive coordination and also long-term memory were damaged. In the \([\text{↑CS}]\) group impairment of long-term memory, a mild short-term memory deficit and also a change of spatial behavior were observed. Additionally, in this group were found the changes in the volume of the whole hippocampus and overturn of right-left volume dominance (laterality) of hippocampus, which were not caused by loss of neurons.

In our experiments, CRH itself affected the cognitively more demanding task (AAPA), mainly by increasing the anxiety of the animals. CRH in combination with corticosterone caused deeper and more permanent disability in cognitively more demanding tasks (AAPA). Corticosterone itself probably influenced mainly memory eventually motivational component which
modulated power and importance of memory traces. This impairment was based apparently on damage of the function and structure of the hippocampus, which was observed after the experiment.

It seems that memory traces created with only a short time in the influence of corticosteroids still intact hippocampal neuroplasticity were sufficiently preserved and persisted longer than the memory traces incurred during or after prolonged exposure to higher levels of corticosteroids.

The period between the second and third weeks of corticosterone administration was critical for normal development and fixing of memory traces in the group with elevated corticosterone (↑CS). For groups with increased corticoliberin (↑CRH and ↑CRH↑CS) were the first signs of behavioral disturbances observed between the second and third weeks of application, but might occur earlier. For these two groups from the results was not possible to determine it, because in this time no behavioral tests were performed.

From our experiments implies that CRH and glucocorticoids affect behavior to some extent in antagonistic manner. While the preponderance of corticoliberin acts and deeps anxiosity, in contrary excess of glucocorticoids induces tranquility. The effect of the relation between these two simultaneously acting on the brain hormones can be applied to modulate behavioral expression. The combined influence of both hormones, is not the sum or product of their isolated effects, but it has another, different quality.