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THE NEUROPROTECTIVE POTENTIALLY OF AGOMELATINE - ANIMAL MODEL STUDY

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Background: Major depressive disorder associated the increase of cortisol level and high level of cortisolemia is correlated with the decrease of neuroprotection. Hippocampus low volume is a marker for depressive disorder. The cognitive impairment in depression is linked with hippocampus - enthorinal structures-frontal cortex disconectivity. Melatoninic agonists can increase the neuroprotection and improve depressive mood.
Methods: We formed 2 study lots each constitued of 5 male adults rats (200-250g), held through the study duration in temperature, humidity, food and ambient stress less conditions, compared to a control lot. The studied substances were administrated intraperitoneal, daily, for 10 days, saline solution equivalent to dexametasone (0.20mg/kg/day) (N1); 14 days we administered 10mg/kg/daily of agomelatine through a stomach tube and the following 10 days of dexametasone and agomelatine in the same dosage (N2). The N1 lot was sacrificed in the day 11, and the N2 lot was sacrificed in the day 25.
The sample brain was histopathologically processed: formalin (10%) and ethyl alcohol (96%) fixation and paraffine embeded. Microtome slices were stained in hematoxyline-eosine, trichromicGS, PAS-hematoxyline, toluidine blue and methylen blue for Nissle corpuscles. The obtained slices were studied with optical microscopy, the target of our study was hippocampus and frontal cortex.
Results: The hippocampus and frontal cortex presents important histopathological abnormalities (neuronal death, vacuolisation, axonal fragmentation) for N1 after dexametasone. These changes are decreased in N2.
Conclusions: Agomelatine on the animal model actioned with a neuroprotective effect in the hippocampus and frontal cortex front of cortisolic aggresion (marker for depression).

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