

SCNS. 13. Effect of Riparins I e III, isolated from *Aniba riparia*, on corticosteroneinduced recent memory impairment in mice

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Introduction: Exposure to stress in mice and psychosocial stress in humans can alter cognitive functions such as learning and memory, and also be associated with the physiopathology of mood disorders and anxiety. Anxiolytic agents and sedatives nowadays prescribed for the treatment of such illnesses do not cause improvement in cognitive function, and even worsening it, as the benzodiazepines do. In previous studies, Riparins I and III (Ripl and RiplII), isolated from Aniba riparia, showed themselves to be affective in animal models predictive of anxiolytic and antidepressant activities, not causing change in hippocratic models. **Objectives:** Therefore, considering the potential of natural products to the isolation of new drugs, the goal of the present work was to investigate the aspects of Ripl and RiplII in the post-stress memory dysfunction caused by repeated injections of corticosterone (CORT). Methods: For that, were used Swiss female mice, weight between 22-25g, divided according to the following experimental groups: Control group (vehicle saline, 1% de tween80, 1% de DMSO, s.c., for 14 and 21 consecutive days), Stressed group (CORT, 20mg/kg, s.c, for 14 or 21 days), Group treated with Ripl or III (50 mg/kg, orally for 8 days), Group treated with fluvoxamine (Flu 50 mg/kg, orally, for 8 days). Treatments occurred beginning the 14th day of corticosterone-induced stress and remained simultaneously with it. Depression was induced with several injections of CORT (20mg/kg. s.c.) beyond the period of 14 days. Behavior models analyzed were open field test and passive avoidance test. Administration of Rips recovered the recent memory of animals to the same level as the control group's (Cont: 249.7 ± 24.87 ; Cort: 79.00 ± 45.68 ; Ripl: 300 ± 0 ; RipIII: 293.6 \pm 6.429; Flu: 300.0 \pm 0; p < 0.001). **Conclusions:** The findings indicate that the repeated administration of Ripl and RiplII recovered the memory loss induced by stress and corticosterone in mice.

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