Participation of the endocannabinoid system in stress-induced impairment of procedural memory acquisition

Poster

Comportamiento/Cognitivo - Behavioral/Cognitive

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Stress is a physiological response characterized by the release of glucocorticoids (GC), mainly corticosterone (CO) in rodents. Stress modulates memory depending on the phase (acquisition or retention) and type of memory involved. Procedural memory, associated with skills, depends on the dorsal striatum (DS) and is affected by stress; restraint stress (R) or intraperitoneal (ip) CO impair acquisition [1], whereas ip CO enhances consolidation [2]. In addition, GC effects on consolidation involve the endocannabinoid system (ECS) [3]. Therefore, this study evaluated the effects of R or CO before training, in the elevated T maze and whether GC recruit the ECS to modulate procedural memory acquisition. Male Wistar rats implanted with cannulae in the DS were assigned to independent groups: VEH (0.1% DMSO), R (15 min of R+ intrastriatal 0.1% DMSO), CO (5 mg/kg, ip 40 min before+ intrastriatal 0.1%DMSO), WIN55212 (CB1 agonists 10 µM) with or without stressors (WIN+R, WIN+CO, WIN), or AM251 (CB1 antagonist,1 µM) with or without stressors (AM+R, AM+CO, AM), CO-DS (intrastriatal CO, 10 µM, 30 min before) or AM+CO-DS. Drugs were administered 5 min before each stressor and then trained in the ETM. Retention was evaluated 24 h later. Activation of CB1 receptors impaired acquisition and retention similarly to R and CO. In contrast, AM251 prevented the impairment induced by both stressors. Likewise, CO-DS impaired memory, this effect was blocked by AM251. These findings suggest that

GC recruit the ECS to mediate procedural memory impairment during acquisition. The distinct effects of stress may involve other cellular mechanisms.

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