Amygdale dysfunction as a possible neural basis for stress-induced alterations in nociception and cognitive functions: Implication of NMDA receptors

M. Nazeri1, M. Shabani2, F. Abareghi1,*, M. Kermani1, L. Golchin3, S. Parsania3

1Medical student’s research committee, Kerman University of medical science, Kerman, Iran
2Intracellular Recording Lab, Neuroscience Research center, Kerman Medical University, Kerman, Iran
3Neuroscience Research center, Kerman Medical University, Kerman, Iran

Background and Aims: Chronic stress is considered as one of the contributors to chronic pain. Effect of chronic swim stress on nociception and passive avoidance task (PA) was evaluated in this study. Role of NMDA receptors was evaluated by administering Ketamine (NMDAR antagonist) before each swimming session.

Methods: Male wistar rats were used for this study. Baseline measurements were recorded for nociception. Nociception was evaluated using hot-plate (53.5°C) and tail flick test and PA was evaluated using Shuttle-box instrument. Ketamine (5mg/kg, i.p.) or saline were administered to rats before each swimming session. Rats were exposed to swimming stress in 50cm height of water in a 20min session for 3 consecutive days. Nociception and PA were evaluated in the day 4. Data was analyzed using paired t-test and student t-test. P<0.05 was considered statistically significant.

Results: Rats in FS group showed a decreased reaction time in hot-pate and tail-flick test (p<0.01), besides an impaired learning and memory in PA (a decreased Step-Through Latency (STL)) (p<0.05). Administration of ketamine (5mg/kg, i.p.) reversed this decrease in pain threshold, and memory impairments were also reversed by ketamine, while learning impairment was not changed by ketamine. Rats receiving saline prior to each simming session did not show any difference in nociception and PA.

Conclusions: This study showed that PA, simultaneous with nociception, is impaired after chronic swim stress, implicating a role for amygdale dysfunction. Antagonism of NMDAR reversed this effect of chronic stress, demonstrating an implication for Glutamergic neurotransmission in hyperalgesia and memory impairments following chronic swim stress. These findings suggest a novel mechanism for stress-induced hyperalgesia and psychophysical stress-induced amnesia, and amygdale seems to be a crucial brain region in these two phenomenon.

Keywords: Stress-induced hyperalgesia; Stress-induced amnesia; Amygdale; NMDA receptors