P1822 / #2736

Topic: AS13 Emotion, Memory and Cognition

CHRONIC SOCIAL DEFEAT STRESS INDUCES ANXIETY AND MEMORY IMPAIRMENT IN MALE MICE: ROLE OF THE BNST, AMYGDALA, AND HIPPOCAMPUS

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Chronic exposure to social defeat stress (SDS) induces anxiety-like responses and memory impairment. In this study, we hypothesize that these emotional, and cognitive consequences induced by SDS are modulated by glutamatergic neurons located in the bed nucleus of the stria terminalis (BNST), amygdaloid complex, and hippocampus in mice. Thus, we investigated the influence of chronic SDS on (i) the avoidance behavior assessed in the social interaction test, (ii) the anxiety-like behavior (e.g., elevated plus-maze, and open field tests) (iii) the short-term memory (object recognition test), (iv) ΔFosB, CaMKII as well as ΔFosB + CaMKII labeling in neurons located in the BNST, amygdaloid complex, dorsal (dHPC) and the ventral (vHPC) hippocampus in male mice. Results showed that SDS (a) increased defensive and anxiety-like behaviors and led to memory impairment; (b) increased ΔFosB + CaMKII labeling in BNST and amygdala, suggesting that both areas are strongly involved in the modulation of this type of stress; and increased and decreased ΔFosB labeling in the vHPC and dHPC, respectively, suggesting that the vHPC is likely related to the increase of defensive- and anxiety-related behaviors, whereas the dHPC seems to modulate the memory impairment. Present findings indicate the involvement of glutamatergic neurotransmission in the circuits that modulate emotional and cognitive consequences induced by social defeat stress.

Declaration of Interest Statement: None

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METHYL JASMONATE RESCUES STRUCTURAL ALTERATIONS IN CORTICOLIMBIC NEURONS PRODUCED BY THE UNPREDICTABLE CHRONIC MILD STRESS MODEL OF DEPRESSION

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The unpredictable chronic mild stress (UCMS) mouse model of depression causes a variety of neuronal structural alterations ranging from dendritic spines loss and dendrite arbor retraction to spine proliferation in brain regions involved in the control of mood. Based on data showing that the plant stress hormone methyl jasmonate (MJ: methyl acetate-2,2-d2) mimics the effect of imipramine in rescuing the UCMS-induced depressive behavioral phenotype, here we examine whether the UCMS protocol used to demonstrate the antidepressant action of the compound triggers structural alterations in the basolateral amygdala, hippocampus and prefrontal cortex which can be rescued by MJ treatment. Male C57BL/6 mice were injected with MJ (50 mg/kg) or saline (SAL) before each of the two daily exposures to UCMS and administered over 10 days. Home cage, daily manipulated, SAL-injected mice served as controls (CTRL). On day 11, mice were sacrificed for Golgi staining. Results show that in comparison with CTRL mice, SAL + UCMS mice exhibit a massive reduction in spine density and dendritic arbor extension/complexity in the three regions examined. Remarkably, MJ + UCMS mice show an alleviation of neuronal structural alterations in the three regions, with a more complete recovery observed in the prefrontal cortex. Our data provide further validation of the antidepressant action of the compound by revealing its efficacy in globally preventing the collapse in synaptic density and neuronal connectivity identified as key nodes of the mood circuitry.

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MALADAPTIVE BEHAVIOURS AND HISTOARCHITECHTURAL DERANGEMENT OF WISTAR RATS' BRAIN FOLLOWING ORAL ADMINISTRATION OF SUBSTANCE/DRUG (DRUG ABUSE)

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Drug/substance abuse proliferation especially among teenagers are on the alarming rate. The neurobehavioral changes associated with over dependence on substance/drug ranges from mild to severe even fatal consequences. The effect of substance/drug (alcohol, marijuana and tramadol) abuse on neurobehavioral pattern and the histoarchitecture of Wistar rats' brain were evaluated in this study. A total of 20 rats were procured from the Physiology Department Animal House, acclimatized for 7 days and group into 4 of 5 rats each, fed with normal rat chow. Group A received normal rat chow and water albinitum. Group B received 1 ml/mg alcohol while Group C and D received 2 ml/mg Marijuana and Tramadol for 21 days respectively. Neurobehavioural changes were assessed using Beam walking, open field maze and Morris water maze while the variation in the histoarchitecture of some regions were ascertained using histological staining and imaging using high resolution custom built microscope. The result showed that the test groups were significantly very sluggish in movement using the line crossing in both beam walking (38.75 ± 0.85, 38.67 ± 0.76, 26.25 ± 1.61 and 31.4 ± 1.47) and open field maze (59.75 ± 1.25, 42 ± 3.04, 44.6 ± 2.08 and 46 ± 2.12) for control, alcohol, marijuana and tramadol respectively. Other parameters varied significantly too. The result also showed that substance/drug abuse affects learning and memory. The visible platform results were significantly higher in the test groups than the control (11.57 ± 0.4, 20.57 ± 0.5, 23.54 ± 0.9 and 28.28 ±). The histological images excessive vacoulation in the cerebral cortex, hippocampus and cerebellum among the test groups especially at higher magnification. The study established that maladaptive tendencies are possibly due to impairment in the