

**FC03-03 - ACUTE TREATMENT WITH CANNABINOID RECEPTOR AGONIST WIN55212,2 IMPROVES PREPULSE INHIBITION IN PSYCHOSOCIALLY STRESSED MICE**

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Cannabis, similar to psychosocial stress, is well known to exacerbate psychotic experiences and can precipitate psychotic episodes *de novo* in vulnerable individuals. Cannabinoid receptors 1 are widely expressed in the central nervous system (CB1) and are particularly important to mediate the effects of cannabis. Chronic cannabis use in patients and chronic cannabinoids treatment in animals is known to cause reduced prepulse inhibition (PPI). Similarly, chronic psychosocial stress in mice impairs PPI. In the present study, we investigated the synergistic effects of substances modulating the CB1 receptors and chronic psychosocial stress (social defeat). CB1 agonists serve as a surrogate marker for the effects of cannabis on the brain. For this purpose, C57Bl/6J mice were exposed for three weeks to psychosocial stress using the resident-intruder paradigm and were subsequently acutely treated with cannabinoid receptor agonist WIN55212,2 (3 mg/kg, i.p.). Stressed mice displayed a higher vulnerability to WIN55212,2 in the open field and PPI tests. The effects of WIN55212,2 on PPI were antagonized by Rimonabant (SR141716A, 3 mg/kg), a specific CB1 receptors antagonist suggesting an involvement of CB1 receptors in sensorimotor gating. Interestingly, WIN55212,2 increased PPI in psychosocially stressed mice although previous studies in rats showed the opposite effects. Our data may imply that cannabinoids may improve particular surrogate marker of psychotic symptoms under certain conditions like psychosocial stress. However cannabinoids/CB1 receptor agonists may have possibly opposite effects in different experimental animal conditions. Further studies have to be done to study possible factors being involved in the expression of CB1 receptor effects.