GS03-5 EARLY LIFE STRESS INDUCES CHRONIC PAIN IN MICE

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Increasing evidence demonstrates a causal relationship between early life stress and vulnerability or resilience to stress in adult life. Although the mechanisms underlying early life stress-induced vulnerability are unclear, abnormalities of brain function have been observed in psychiatric patients who experienced stress in early life. Stressful events, including early life stress, have negative effects on neuropathic pain. Therefore, early life stress contributes to the pathogenesis of psychiatric disorders and chronic pain in adult patients. Our previous studies demonstrated that maternal separation combined with social isolation exacerbates nerve injury-induced hyperalgesia and sex-dependently increases depression-like behaviors after nerve injury in mice. We found that changes in BDNF expression after early life stress may be associated with neuropathic pain-induced depression-like behavior in adulthood. Furthermore, early life stress sex-dependently and site-specifically increases neuronal activity in the brain. These results suggest that increased neuronal activity in multiple brain regions of mice subjected to early life stress may enhance hyperalgesia after nerve injury. In addition, we found that early life stress-induced activated astrocytes in the LC may contribute to the exacerbation of neuropathic pain. More recently, we found that antinociceptive effects of morphine and morphine-induced hyper-locomotion are reduced in early life stress mice. In this symposium, we summarize the findings of our recent study about the mechanism of early life stress induced chronic pain.