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Reduction of Transient Receptor Potential Vanilloid 1 for Chronic Pain and Depression Co-Morbidity through Electroacupuncture and Gene Deletion in Mice Brain

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Abstract : Chronic pain and depression have an estimated 80% rate of comorbidity with unsatisfactory treatment interventions signifying the importance of developing effective therapeutic interventions for a serious chronic condition affecting a large majority of the global population. Chronic pain is defined as persistent pain presenting for over 3 months. This disease state increases the risk of developing depression in comparison to healthy individuals. In the current study, complete Freund's adjuvant (CFA) was used to induce cell-mediated chronic inflammatory pain in a murine model. Significant mechanical and thermal hyperalgesia was induced, alongside observable depression-like behaviors. These conditions were attenuated through the use of electroacupuncture (EA). Similarly, these effects were also investigated with respect to the transient receptor potential vanilloid 1 (TRPV1), by analyzing the effects of TRPV1 gene deletion on the comorbidity of chronic pain and depression. The expression of the TRPV1 inflammatory response, and related downstream molecules, including protein kinases (PKs), mitogen-activated protein kinase (MAPKs), and transcriptional factors, were significantly reduced in the thalamus, prefrontal cortex (PFC), hippocampus, and periaqueductal gray (PAG) of CFA-treated mice. In addition, phosphorylated N-methyl-D-aspartate (NMDA) receptor 1 was also found to be reduced in the aforementioned areas, suggesting potential application and validity in a clinical setting. Our study determined the prospective therapeutic effects of EA in the treatment of chronic inflammatory pain and depression comorbidity and provides a novel and detailed mechanism underlying EA-mediated analgesia. These findings may be relevant in the utilization of clinical intervention approaches related to chronic pain and depression comorbidity.

Keywords : chronic pain, depression, NMDA, prefrontal cortex, TRPV1
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