Role of Transient Receptor Potential Vanilloid 1 in Electroacupuncture Analgesia on Chronic Inflammatory Pain in Mice

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Abstract: Chronic inflammatory pain results from peripheral tissue injury or local inflammation to increase the release of protons, histamines, adenosine triphosphate, and several proinflammatory cytokines. Transient receptor potential vanilloid 1 (TRPV1) is involved in fibromyalgia, neuropathic, and inflammatory pain; however, its exact mechanisms in chronic inflammatory pain are still unclear. We investigate the analgesic effect of EA by injecting complete Freund's adjuvant (CFA) in the hind paw of mice to induce chronic inflammatory pain (> 14 d). Our results showed that EA significantly reduced chronic mechanical and thermal hyperalgesia in the chronic inflammatory pain model. Chronic mechanical and thermal hyperalgesia was also abolished in TRPV1-/- mice. TRPV1 increased in the dorsal root ganglion (DRG) and spinal cord (SC) at 2 weeks after CFA injection. The expression levels of downstream molecules such as pPKA, pPI3K, and pPKC increased, as did those of pERK, pp38, and pJNK. Transcription factors (pCREB and pNFkB) and nociceptive ion channels (Nav1.7 and Nav1.8) were involved in this process. Inflammatory mediators such as GFAP (Glial fibrillary acidic protein), S100B, and RAGE (Receptor for advanced glycation endproducts) were also involved. The expression levels of these molecules were reduced in EA (electroacupuncture) and TRPV1-/-mice but not in the sham EA group. The present study demonstrated that EA or TRPV1 gene deletion reduced chronic inflammatory pain through TRPV1 and related molecules. In addition, our data provided evidence to support the clinical use of EA for treating chronic inflammatory pain.

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