THE ROLE OF TRANSIENT RECEPTOR POTENTIAL VANILLOID 1 AT HYPOTHALAMUS OF MICE IN ACUPUNCTURE RELIEVE CHRONIC PAIN

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BACKGROUND AND AIM

Recently, evidence intimated that the ligand-gated non-selective cation channel TRPV1 is essential in developing inflammatory hyperalgesia channel (1). The pain signaling pathway transmitting from the nociceptive activated by noxious stimulation is reported to be associated with TRPV1 activation and related molecules (2). The transient receptor potential vanilloid 1 has been identified in the CNS and PNS (3). Previous studies indicated that TRPV1 was notably expressed in the hypothalamus (4). The mechanism underlying acupuncture's analgesic efficacy was the inhibition of TRPV1 activation, which inhibits pain signaling to the brain (5). This study aims to investigate the anti-nociception mechanism of electroacupuncture at Zusanli (ST36) acupoint on the expression of TRPV1 in an ICS-induced chronic pain model.

METHODS AND MATERIALS

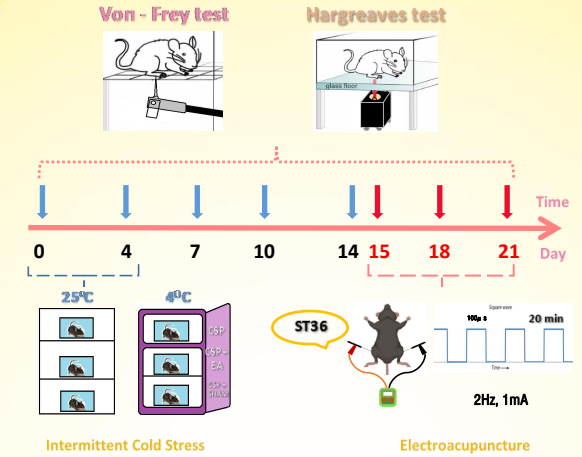


Figure 1: We induced chronic pain in mice by performing an Intermittent cold stress model and examined the mice's behavioral sensitivity by the Von-Frey test and Hargreaves test. EA at ST36 acupoint was inserted at a low frequency of 2 Hz. Furthermore, we observed TRPV1 expression in the hypothalamus.

RESULTS

EA at the ST36 acupoint attenuated nociceptor behavior in mice ICS model

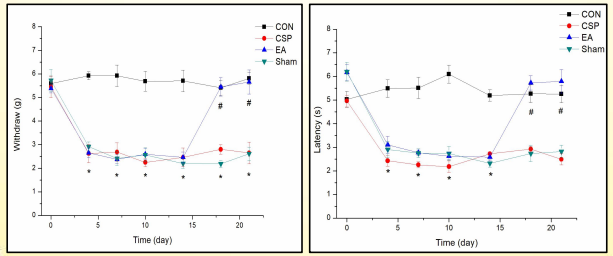
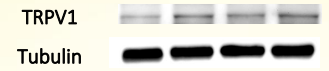


Figure 2: Chronic pain-induced mechanical and thermal hyperalgesia through the ICS model. (a) Von-Frey filament test; (b) Hargreaves test. *p<0.05, as compared to that of the baseline, #p<0.05, comparison between chronic pain and EA-ST36 groups. CSP=Cold stress pain.

EA at the ST36 alleviated overexpression of TRPV1 in hypothalamus tissue by using Western blot



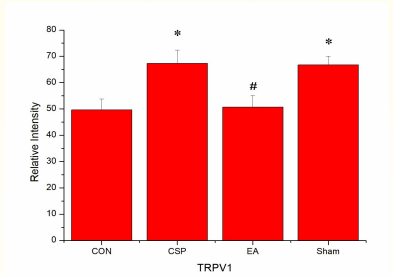


Figure 3: TRPV1 protein level was increased in the hypothalamus in intermittent cold stress-induced chronic pain and further attenuated by EA at the ST36 acupoint in mice. Hypothalamus lysates were immunoreactive with specific antibodies to TRPV1 and significantly increased signal as compared to that of the Control group, and reduced by EA at ST36 acupoint as compared to that of the CSP-induced group, but not altered in the Sham group.

CONCLUSION

We showed that EA at Zusanli (ST36) acupoint at 2Hz low frequency stimulation diminished animal chronic pain behaviour and accompanied by decreasing the expression of TRPV1 in Hypothalamus.

DISCUSSION

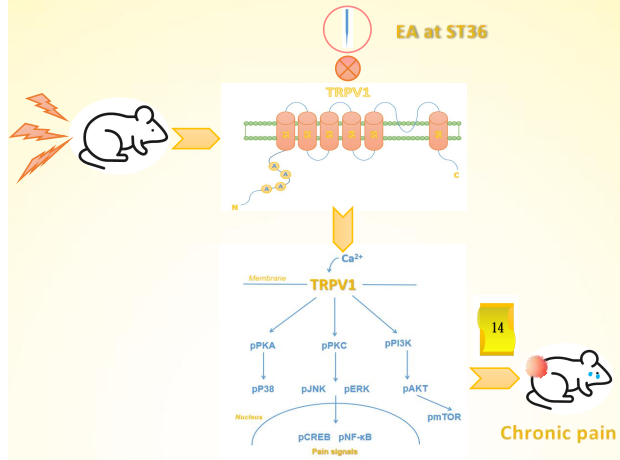


Figure 4: Present research demonstrated the role of TRPV1 in the mechanism of activating the pain signaling pathway in the mice hypothalamus by authorizing the influx of cations. Low-frequency electroacupuncture at ST36 precluded the latter stream from the entrance, thus mitigating chronic pain.

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