BMC Complementary & Alternative Medicine

ORAL PRESENTATION Open Access

OA08.03. Electroacupuncture alleviates hyperalgesia by inhibiting spinal interleukin-17 in an inflammatory pain rat model

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From International Research Congress on Integrative Medicine and Health 2012
Portland, Oregon, USA. 15-18 May 2012

Purpose

Previous studies demonstrated that electroacupuncture (EA) alleviates hyperalgesia, but the mechanisms remained unclear. Because it is well known that interleukin-17 (IL-17) is associated with autoimmune disorders, the present study was designed to determine whether spinal IL-17 plays a role in inflammatory pain and, if so, whether EA inhibits spinal IL-17 expression during such pain.

Methods

Hyperalgesia was induced by injecting complete Freund's adjuvant (CFA, 0.08 ml, 40 μg Mycobacterium tuberculosis) into one hind paw of each rat. EA treatment, 10 Hz at 3 mA, was given at acupoint GB30 twice for 20 min each, once immediately post-CFA and again 2 hours later. Paw withdrawal latency (PWL) was tested before (-48 h) and 2 and 24 hours after CFA to assess behavioral hyperalgesia. IL-17 antibody (0.2-2 μg/rat) was given intrathecally (i.t.) 24 h before CFA to block the action of basal IL-17 and 2 hours prior to each of two PWL tests to block CFA-induced IL-17. I.t. recombinant IL-17 (10-400 ng/rat) was administered to naive rats to determine its effects on PWL and phosphorylation of NR1 (p-NR1). P-NR1 is known to modulate N-methyl-D-aspartate receptor (NMDAR) activity and to facilitate pain. Spinal cords were removed for immunostaining of IL-17, double immunostaining of IL-17/cell markers and IL-17 receptor subtype A (IL-17RA)/NR1, and western blot to measure p-NR1 and IL-17RA.

Results

The data showed that (1) IL-17 is selectively up-regulated in astrocytes, 2) IL-17RA is localized and up-regulated in NR1-immunoreactive neurons, and 3) an IL-17 antibody at 2 μg/rat significantly increased PWL ($p<0.05$) and decreased p-NR1 and IL-17RA in CFA- and IL-17-injected rats compared to control. EA significantly inhibited hyperalgesia, IL-17, IL-17RA, and p-NR1.

Conclusion

The results suggest that (1) spinal IL-17 is produced by astrocytes and enhances p-NR1 to facilitate inflammatory pain, and 2) EA inhibits hyperalgesia by suppressing IL-17.

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Published: 12 June 2012

doi:10.1186/1472-6882-12-S1-O31
Cite this article as: Zhang *et al.*: OA08.03. Electroacupuncture alleviates hyperalgesia by inhibiting spinal interleukin-17 in an inflammatory pain rat model. *BMC Complementary and Alternative Medicine* 2012 **12**(Suppl 1): O31.

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