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ANALGESIC EFFICACY AND MODE OF ACTION OF ELECTROACUPUNCTURE IN A RAT MODEL OF PROSTATE CANCER-INDUCED BONE PAIN

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Introduction: The pathobiology of prostate cancer (PCa)-induced bone pain (PCIBP) has both inflammatory and neuropathic components. We assessed the analgesic efficacy and mode of action of the electroacupuncture (EA) at SP6 and ST36 in a rat model of PCIBP.

Methods: Male Wistar Han rats (100-140 g) were used in this study. At 14-21 days after unilateral intratibial injection of AT3B PCa cells, rats exhibiting hindpaw hypersensitivity were randomly assigned to three groups: control, EA or non-acupoint treated (NA) group. Rats in EA group received EA stimulation at bilateral ST36 and SP6 with 100 Hz once a day for 30 min, those in NA group received EA stimulation at bilateral non-acupoints, and those in control group didn't receive EA stimulation. Three hours after EA stimulation, analgesic efficacy was assessed. The mode of action was investigated using immunohistochemical, Western blot, and/or molecular biological methods in lumbar dorsal root ganglia (DRGs) removed from rats.

Results: EA stimulation at ST36 and SP6 acupoints showed analgesic effect in PCIBP rats. Lumbar DRG levels of angiotensin II, nerve growth factor (NGF), tyrosine kinase A (TrkA), phospho-p38 mitogen-activated protein kinase (MAPK), and phospho-p44/p42 MAPK, but not the AT2 R, were increased significantly ($P < 0.05$) in PCIBP rats. EA produced analgesia in PCIBP rats by reducing elevated angiotensin II levels in the lumbar DRGs to attenuate augmented angiotensin II/AT2 R signaling. This in turn reduced augmented NGF/TrkA signaling in the lumbar DRGs. The net result was inhibition of p38 MAPK and p44/p42 MAPK activation.

Conclusion: EA can relieve the symptoms of intractable PCIBP and other pain types where hyperalgesia worsens symptoms.