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LATE-BREAKING POSTER SESSION III

INHIBITION OF SPINAL ENDOTHELIN TYPE A RECEPTOR AMELIORATES NEUROPATHIC PAIN THROUGH DOWN-REGULATION OF MAPK AND NF-KAPPAB P65 IN RATS

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Background and aims

Endothelin type A receptor (ETAR) plays a critical role in Endothelin-1 (ET-1)-mediated neuropathic pain, a disease in which the mitogen activated protein kinases (MAPK) family and nuclear factor NF- κ B p65 signaling take important parts. Results of our past study showed that inhibition of spinal ETAR alleviates neuropathic pain, but the underlying mechanism remains unknown. Therefore, we hypothesized that inhibition of central ETAR ameliorates neuropathic pain through modulation of the MAPK family and NF- κ B p65.

Methods

Rats were subjected to sciatic nerve ligation (SNL) or sham operation with or without ETAR antagonist, BQ-123, administered intrathecally via implanted catheter at dosages 30 μ g, 60 μ g, or 90 μ g daily for 3 days respectively. Mechanical allodynia was assessed daily 30 minutes before-/after-injection, 1 hour after-injection of BQ-123 from post-SNL day 4 to 6, and once on day 7 (without BQ-123 administration) before rats were sacrificed.

Results

SNL sensitized animal's pain response to *von frey* filaments applied to the ipsilateral hind paw. This observed mechanical allodynia was reduced by inhibition of spinal ETAR at all dosages significantly (*P*

Conclusions

Spinal ETAR inhibition alleviated neuropathic pain in part through inactivation of MAPK family and NF- κ B p65.