The Effect of Epidural Resiniferatoxin in the Neuropathic Pain Rat Model Randomized Trial: A Complementary Mechanism

To the Editor:

I read with great interest the paper by Lee et al titled "The Effect of Epidural Resiniferatoxin in the Neuropathic Pain Rat Model" (1). This work addressed that resiniferatoxin, which is a decoy receptor of transient receptor potential Vanilloid subtype 1, improved neuropathic pain. I would like to complete the discussion of Lee and colleagues by introducing a major complementary route which resiniferatoxin could reduce neuropathic pain.

The essential role of neuropeptides in mediating neuropathic pain is evidenced by many works (2,3). Recent studies have shown that resiniferatoxin not only produces various antinociception factors, including GMAP (galanin message-associated protein) and nitric oxide, but also is capable of down-regulation of neuropeptides such as substance *P* (4,5). Therefore, these important mechanisms should be borne in mind as the major complementary mechanisms for resiniferatoxin – reduced neuropathic pain.

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