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Implications and mechanism of action of gabapentin in neuropathic pain.

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Abstract

Gabapentin is an anti-epileptic agent but now it is also recommended as first line agent in neuropathic pain, particularly in diabetic neuropathy and post herpetic neuralgia. $\alpha 2\delta$ -1, an auxillary subunit of voltage gated calcium channels, has been documented as its main target and its specific binding to this subunit is described to produce different actions responsible for pain attenuation. The binding to $\alpha 2\delta$ -1 subunits inhibits nerve injury-induced trafficking of $\alpha 1$ pore forming units of calcium channels (particularly N-type) from cytoplasm to plasma membrane (membrane trafficking) of pre-synaptic terminals of dorsal root ganglion (DRG) neurons and dorsal horn neurons. Furthermore, the axoplasmic transport of $\alpha 2\delta$ -1 subunits from DRG to dorsal horns neurons in the form of anterograde trafficking is also inhibited in response to gabapentin administration. Gabapentin has also been shown to induce modulate other targets including transient receptor potential channels, NMDA receptors, protein kinase C and inflammatory cytokines. It may also act on supra-spinal region to stimulate noradrenaline mediated descending inhibition, which contributes to its anti-hypersensitivity action in neuropathic pain.

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