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Poster Sessions

Implicated in spinal cholecystokinin-8 and hemokinin-1 in type 1 diabetes allodynia in mice

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Diabetes mellitus is one of the major causes of peripheral neuropathy which represented by the mechanical allodynia. The mechanical allodynia has been suggested to involve several pain-related neurotransmitters in the dorsal spinal cord. The sulfated octapeptide cholecystokinin (CCK-8) is present in the dorsal spinal cord in mice, which is involved in modulating afferent nociceptive information. Hemokinin-1, a kind of tachykinin family, is present in the spinal cord, which effects on the tachykinin neurokinin 1 (NK₁) receptor just like substance P and is involved in the pain transmission. Our study was designed to determine a potential involvement of spinal CCK-8 and hemokinin-1 in mediating streptozotocin (STZ)-induced type 1 diabetes allodynia in mice. STZ (200 mg/kg, i.v.)-induced mechanical allodynia was evoked significantly 7 day. The mechanical allodynia elicited by STZ was inhibited by intrathecal (i.t.) administration of antiserum against CCK-8 and hemokinin-1, and CI-988, a CCK-B receptor antagonist. No significant of mechanical allodynia induced by STZ was shown in i.t. administration of SR-27897, a CCK-A receptor antagonist. The level of CCK receptors mRNA in the dorsal root ganglia and spinal dorsal horn of the STZ-induced type 1 diabetes mice was measured by real time PCR. The mRNA level of CCK-B receptors increased significantly in STZ-induced diabetic allodynia in mice. The present results suggest that the STZ-induced mechanical allodynia are mediated through the spinal CCK-8 and hemokinin-1.