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Treatment with anti-interleukin-6 receptor antibody improves mechanical hypersensitivity and cognitive dysfunction in mice with neuropathic pain

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Neuropathic pain is frequently accompanied by cognitive dysfunction, yet the mechanisms remain unclear. Interleukin-6 (IL-6) plays an important role in both chronic pain and cognitive impairment. We observed that IL-6 was increased in spinal dorsal horn and hippocampus of mice with neuropathic pain. The current study examined whether IL-6 blockade by a monoclonal anti-IL-6 receptor antibody (MR16-1) improves mechanical allodynia and cognitive dysfunction in mice with neuropathic pain. Neuropathic pain was induced by partial sciatic nerve ligation (PSNL) of male ddY mice. Novel object recognition test was performed to evaluate cognitive function. Mechanical threshold was determined using von Frey filaments. PSNL induced mechanical allodynia at 1 week and 2 weeks post-injury. Cognitive dysfunction was observed at 2 weeks but not 1 week after PSNL. Intraperitoneal injection of MR16-1 improved mechanical allodynia, but not cognitive dysfunction. On the other hand, intranasal administration of MR16-1 improved cognitive dysfunction, but not mechanical allodynia. Together, the current study suggests that IL-6 signaling could be involved in both of mechanical allodynia and cognitive dysfunction in neuropathic pain of mice. Furthermore, IL-6 might have distinct roles in the regulation of nociceptive transduction and cognitive function.