研究業績　英文表記

和文	
表題	不動化による知覚過敏における側坐核のドーパミン神経伝達調節障害
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英文	
Title	Dysregulation of dopamine neurotransmission in the nucleus accumbens in immobilization-induced hypersensitivity
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Abstract	Cast immobilization causes sensory hypersensitivity, which is also a symptom of neuropathic pain and chronic pain. However, the mechanisms underlying immobilization-induced hypersensitivity remain unclear. The present study investigated the role of dopamine neurotransmission in the nucleus accumbens shell (NAcSh) of rats with cast immobilization-induced mechanical hypersensitivity using in vivo microdialysis. Cast immobilization of the hind limb decreased the paw withdrawal threshold (PWT). Mechanical stimulation of the cast-immobilized hind limb induced a decrease in dopamine in the NAcSh, and this decrease was associated with the upregulation of presynaptic D2-like receptors. A D2-like receptor antagonist infused into the NAcSh reversed the decrease in PWT in rats with cast immobilization, whereas a D2-like receptor agonist infused into the NAcSh induced a decrease in PWT in control rats. In addition, the expression of the D2 receptor (Drd2) mRNA in the NAcSh was increased by cast immobilization. Importantly, systemic administration of the D2-like receptor antagonist reversed the decrease in PWT in rats with cast immobilization. As dopamine levels regulated by presynaptic D2-like receptors did not correlate with the PWT, it is presumed that the D2-like receptor antagonist or agonist acts on postsynaptic D2-like receptors. These results suggest that immobilization-induced mechanical hypersensitivity is attributable to the upregulation of postsynaptic D2-like receptors in the NAc. Blockade of D2-like receptors in the NAcSh is a potential therapeutic strategy for immobilization-induced hypersensitivity.
keyword	D2-like receptor; hyperalgesia; microdialysis; postsynaptic; rat.

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