Neuropathic and inflammatory pain increases glutamatergic excitatory postsynaptic current on adult rat spinal dorsal horn neurons.

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Background; Chronic pain is characterized by abnormal sensitivity to normal stimulation coupled with a feeling of unpleasantness. This condition afflicts people worldwide and severely impacts their quality of life and has become an escalating health problem. Two major models are used to study chronic pain in animals, including nerve injury and the injection of a complete Freund's adjuvant (CFA) into the hind paw. However, how these models induce glutamatergic synaptic plasticity in the spinal cord is not fully understood.

Methods; Using *in vitro* and *in vivo* whole-cell patch-clamp recording methods, we analyzed spontaneous excitatory postsynaptic currents (sEPSCs) 2 weeks following nerve injury and 1 week following CFA injection.

Results; In the spinal slice preparation, these models increased both the frequency and amplitude of sEPSCs in SG neurons. Next, we analyzed the active electrophysiological properties of neurons, which included; resting membrane potentials (RMPs) and the generation of action potentials (APs) *in vitro*. Interestingly, about 20% of recorded SG neurons in this group elicited spontaneous APs (sAPs) without changing the RMPs. Furthermore, we performed *in vivo* whole-cell patch-clamp recording in SG neurons to analyze active electrophysiological properties under physiological conditions. Importantly, *in vivo* SG neurons generated sAPs without affecting RMP in the nerve injury and the CFA group.

Conclusions; Our study describes how animal models of chronic pain influence both passive and active electrophysiological properties of spinal SG neurons.