Mechanism of bilateral pain under the inflammatory state

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The unilateral peripheral injury or inflammation produces in bilateral hypersensitivity to pain in the ipsilateral and contralateral sides. This phenomenon is reported in clinical pain syndromes and in various animal pain models. However, its mechanism is not cleared. In the present study, we investigated the mechanism of this phenomenon using the inflammatory pain model and the capsaicin test. Chronic inflammatory pain was induced by injecting the complete Freund's adjuvant (CFA) and assessed the mechanical allodynia by von Frey filament test. After CFA injection, the paw withdrawal threshold in CFA injected paw reached a minimum at 3 days after CFA injection. Furthermore, in mice pretreated with CFA in the left hindpaw 3 days before, injection of low-dose (this dose did not produce significant pain-related behaviors) capsaicin in the right hindpaw induced the remarkable pain-related behaviors against capsaicin non-injected paw. This phenomenon was attenuated by the administration of $TRPV_1$receptor antagonist. Moreover, bilateral increase of the mRNA expression level of spinal $TRPV_1$receptor was observed at 3 days after CFA pretreatment. These results suggest that the bilateral activation of $TRPV_1$receptor under the inflammatory state induced capsaicin related pain in non-injected side.

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