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The role of TRPV1 channels in carrageenan-induced mechanical hyperalgesia in mice  
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#### Abstract

(Objective) Peripheral inflammation leads to ipsilateral and contralateral mechanical hyperalgesia. The transient receptor potential channel vanilloid type 1 (TRPV1), a nonselective cation channel expressed in mammalian primary sensory neurons and the spinal cord, may be involved in peripheral inflammation, but there is no consensus on the role of this channel in inflammation-induced mechanical hyperalgesia [1,11,12].

(Methods) We examined the role of TRPV1 channels in carrageenan-induced mechanical hyperalgesia using wild-type and TRPV1-knockout (KO) mice and compared the results with those obtained in mice peripherally administered capsaizepine, a TRPV1 antagonist, or capsaicin, a TRPV1 agonist [13,14,15,16,17].

(Results) In the TRPV1-KO mice, ipsilateral mechanical hyperalgesia was significantly reduced during the acute phase (10–60 min), and the contralateral mechanical hyperalgesia nearly disappeared during both the acute and subacute phases. Blocking peripheral TRPV1 using capsaizepine before carrageenan administration resulted in similar effects as those observed in the TRPV1-KO mice, except that it was less effective against contralateral mechanical hyperalgesia during the subacute phase. In contrast, capsaicin remarkably decreased ipsilateral and contralateral mechanical hyperalgesia throughout both phases, but this analgesic effect was not observed in the TRPV1-KO mice.

(Conclusions) TRPV1 channels could be involved in the development of both ipsilateral and contralateral mechanical hyperalgesia after inflammation. Peripheral TRPV1 could participate in acute hyperalgesia, whereas central TRPV1 may participate in subacute secondary hyperalgesia. Capsaicin potentially acts on both primary and secondary hyperalgesia in a TRPV1-dependent manner.