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Poster Sessions

Involvement of Ca_v3.2 T-type channel in inflammatory pain via an interaction with nociceptive TRPA1.

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Low voltage-activated Ca²⁺ channel (Ca_v3.2) and TRPA1 play a key role in inflammatory and neuropathic pain. We previously reported the functional interaction between Ca_v3.2 and TRPA1. However, little is known about the significance of this interaction in pathological conditions. Here, we investigated possible involvement of these channel interactions in inflammatory pain model constructed by intraplantar injection of CFA. At the inflammatory side of DRGs, the protein expression of Ca_v3.2, but not TRPA1 was increased. mRNA of both channels was unchanged by CFA. Depolarizing pulses evoked inward currents which were inhibited by NNC 55-0396, a T-type channel blocker, and were enlarged in the TRPA1-expressing DRG neurons at the inflammatory side. The Ca_v3.2-mediated [Ca²⁺]_i increases were enhanced and the inhibitory rate by HC030031, a TRPA1 blocker, was greater in the TRPA1-expressing DRG neurons at the inflammatory side. In the present study, we showed that the augmentation of [Ca²⁺]_i response to Ca_v3.2 activation may be mediated by the interactions of TRPA1 in addition to the increment of Ca_v3.2 expression under inflammatory conditions. These data suggest that both channels are promising therapeutic targets for inflammatory pain.