TRPV1

- Oral administration of undenatured type II collagen significantly inhibits arthritis-associated pain signal in a mouse model of collagen antibody-induced arthritis and meniscus removal
- The Antinociceptive Effect of Nicorandil in Neuropathic and Nociceptive Pain is Partially Mediated via TRPV1/Opioidergic Signaling
- Human Placental Extract as a Promising Epidural Therapy for Lumbar Spinal Stenosis: Enhancing Axonal Plasticity and Mitigating Pain and Inflammation in a Rat Model
- Dynamic changes in intestinal microbiota mediate mechanical hyperalgesia in surgical menopause model: a potential mechanism of DRG neuroinflammation
- Cyclodextrins inhibit TRPV1 and TRPA1 activation-induced nociception via cholesterol depletion
- Regulatory Action of Calcium and Calcium Channels in Pain Pathways
- New cannabidiol structure-related terpene N-acyl-hydrazones with potent antinociceptive and anti-inflammatory activity
- Ex vivo stimulation of the trigeminal nucleus caudalis induces peripheral CGRP release in the trigeminal ganglion and reveals a distinct dopamine-endocannabinoid mechanism relevant to migraine

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Activated by heat (temperature above 43°C), capsaicin (the pungent compound in chili peppers), and certain endogenous signaling molecules. TRPV1 is primarily expressed in sensory neurons and is involved in the perception of pain and temperature.

The transient receptor potential cation channel subfamily V member 1 (TrpV1), also known as the capsaicin receptor and the vanilloid receptor 1, is a protein that, in humans, is encoded by the TRPV1 gene. It was the first isolated member of the transient receptor potential vanilloid receptor proteins that in turn are a sub-family of the transient receptor potential protein group.

This protein is a member of the TRPV group of transient receptor potential family of ion channels.

The function of TRPV1 is the detection and regulation of body temperature. In addition, TRPV1 provides a sensation of scalding heat and pain (nociception).

TRPV1 is a non-selective calcium channel that is involved in the pathology of neuroinflammation.

NLRP3 inflammasome plays an important role in the development of neuroinflammation after subarachnoid hemorrhage, but the mechanism of NLRP3 inflammasome activation after subarachnoid hemorrhage is still unclear. A study showed that TRPV1 was significantly upregulated after subarachnoid hemorrhage and was predominantly expressed in microglia/macrophages. Antagonism of TRPV1 was effective in ameliorating neurological impairment, brain edema, and neuronal damage, and reducing the inflammatory response (evidenced by reducing the number of CD16/32 positive microglia/macrophages, inhibiting the expression of CD16, CD32, CD86, IL-1b, TNF-a and blocking NLRP3 inflammasome activation). However, this effect can be abolished by NLRP3 inflammasome antagonist MCC950. In vitro experiments confirmed that TRPV1 activated NLRP3 inflammasome by increasing intracellular calcium levels. In conclusion, TRPV1 mediates EBI after SAH via

calcium/NLRP3, and TRPV1 is a potential therapeutic target after SAH [1].

Hong et al. examined histological changes in the DNA methylation within the discs and their association with pain-related transient receptor potential vanilloid subtype 1 (TrpV1) expression in rats subjected to IDD. Epigenetic markers (5-hydroxymethylcytosine (5hmC), 5-methylcytosine (5Mc)), DNA methyltransferases (DNMTs), and Ten-eleven translocations (Tets) were analyzed using immunohistochemistry, real-time PCR, and DNA dot-blot following IDD. Results revealed high 5mC levels in the annulus fibrosus (AF) region within the disc after IDD and an association with TrpV1 expression. DNMT1 is mainly involved in 5mC conversion in degenerated discs. However, 5hmC levels did not differ between groups. A degenerated disc can lead to locomotor defects as assessed by ladder and tail suspension tests, no pain signals in the von Frey test, upregulated matrix metalloproteinase-3, and downregulated aggrecan levels within the disc. Thus, we found that the DNA methylation status in the AF region of the disc was mainly changed after IDD and associated with aberrant TrpV1 expression in degenerated discs [2].

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Zhang K, Qin Z, Chen J, Guo G, Jiang X, Wang F, Zhuang J, Zhang Z. TRPV1 modulated NLRP3 inflammasome activation via calcium in experimental subarachnoid hemorrhage. Aging (Albany NY). 2024 Jan 4;15. doi: 10.18632/aging.205379. Epub ahead of print. PMID: 38180747.
2)
Hong JY, Kim H, Jeon WJ, Lee J, Yeo C, Lee YJ, Ha IH. Epigenetic Changes within the Annulus Fibrosus by DNA Methylation in Rat Intervertebral Disc Degeneration Model. Cells. 2022 Nov 10;11(22):3547. doi: 10.3390/cells11223547. PMID: 36428977.