



Product Information

Anti-Vanilloid Receptor-1 (VR-1)

Developed in Rabbit
IgG Fraction of Antiserum

Product Number **V 2764**

Product Description

Anti-Vanilloid Receptor-1 (VR-1), is developed in rabbit using as immunogen a synthetic peptide corresponding to the C-terminus of rat vanilloid receptor-1 (VR-1), (amino acids 817-838) conjugated to KLH. The sequence is highly conserved in guinea pig (~85%) and human (~70%) vanilloid receptor-1 and is not found in rat VRL-1. Whole antiserum is fractionated and then further purified by ion-exchange chromatography to provide the IgG fraction of antiserum that is essentially free of other rabbit serum proteins.

Anti-Vanilloid Receptor-1 (VR-1) recognizes vanilloid receptor-1 (90 kDa) by immunoblotting. Staining of the vanilloid receptor-1 band in immunoblotting is specifically inhibited with the vanilloid receptor-1 immunizing peptide (rat, amino acids 817-838).

Vanilloid Receptor-1 (VR-1, TRPV1), also known as capsaicin receptor, participates in the sensation of thermal and inflammatory pain.^{1,2} VR-1 responds to multiple noxious stimuli including capsaicin, the pungent main component of "hot" chilli peppers, and moderate (43 °C) thermal stimuli, and is also activated by protons (extracellular acidification), all of which cause pain stimuli *in vivo*. VR-1 is able to integrate simultaneous exposure to both thermal and chemical stimuli.

The VR-1 receptor is a non-selective cation channel structurally related to the TRP family of ion channels. It is highly permeable to divalent cations, including Ca^{2+} and Mg^{2+} , as well as to monovalent cations Na^+ and K^+ . Multiple 5' splice variant (VR.5'sv) have been hypothesized deriving from a common VR gene.³ An additional member of the VRs family is VRL-1/TRPV2, a vanilloid receptor homolog (49% identity to VR-1), activated by high temperatures (>52 °C), but does not respond to capsaicin and low pH.⁴ VR-1 is predicted to contain six transmembrane-spanning segments flanked by large intracellular N-terminal and C-terminal domains, a P-loop membrane domain, and three ankyrin-repeat domains.

Expression of VR-1 appears to be limited to small diameter sensory neurons within the dorsal root ganglia of the spinal cord.^{1,5-6} Multiple signaling pathways modulate VR-1 activity. VR-1 is a component of the PLC signaling pathway in which PLC-coupled receptors increase thermal sensitivity. The pro-inflammatory peptide bradykinin, known to mediate hyperalgesia by stimulating sensory neurons, has been reported to induce VR-1 activity by activating the lipoxygenase pathway of arachidonic acid metabolism as well as a PKC ϵ mediated signaling pathway.^{7,8} VR-1 gene knockout mice show normal responses to noxious mechanical stimuli, but exhibit loss of many of the capsaicin-, acid-, and heat-gated responses in small diameter dorsal root ganglion neurons and also exhibit complete loss of thermal hyperalgesia *in vivo*.^{9,10}

Reagent

Anti-Vanilloid Receptor-1 (VR-1) is supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide.

Precautions and Disclaimer

Due to the sodium azide content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazards and safe handling practices.

Storage/Stability

For continuous use, store at 2-8 °C for up to one month. For prolonged storage, freeze in working aliquots at -20 °C. Repeated freezing and thawing is not recommended. Storage in frost-free freezers is also not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilutions should be discarded if not used within 12 hours.

Product Profile

A minimum working dilution of 1:1,000 is determined by immunoblotting using a whole cell extract of 293T cells expressing rat VR-1.

Note: In order to obtain the best results using various techniques and preparations, we recommend determining the optimal working dilutions by titration.

References

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