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CASR Blocking Peptide (C-term)

Synthetic peptide
Catalog # BP21649b

Specification

CASR Blocking Peptide (C-term) - Product Information

Primary Accession P41180

CASR Blocking Peptide (C-term) - Additional Information

Gene ID 846

Other Names
Extracellular calcium-sensing receptor, CaSR, Parathyroid cell calcium-sensing receptor 1, PCaR1, CASR, GPRC2A, PCAR1

Target/Specificity
The synthetic peptide sequence is selected from aa 1029-1045 of HUMAN CASR

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

CASR Blocking Peptide (C-term) - Protein Information

Name CASR {ECO:0000303|PubMed:16740594, ECO:0000312|HGNC:HGNC:1514}

Function
G-protein-coupled receptor that senses changes in the extracellular concentration of calcium ions and plays a key role in maintaining calcium homeostasis (PubMed:17555508, PubMed:19789209, PubMed:21566075, PubMed:22114145, PubMed:22789683, PubMed:23966241, PubMed:25104082, PubMed:25292184, PubMed:25766501, PubMed:26386835, PubMed:32817431, PubMed:33603117, PubMed:<a

href="http://www.uniprot.org/citations/34194040" target="_blank">34194040, PubMed:34467854, PubMed:7759551, PubMed:8636323, PubMed:8702647, PubMed:8878438). Senses fluctuations in the circulating calcium concentration: activated by elevated circulating calcium, leading to decreased parathyroid hormone (PTH) secretion in parathyroid glands (By similarity). In kidneys, acts as a key regulator of renal tubular calcium resorption (By similarity). Ligand binding causes a conformation change that triggers signaling via guanine nucleotide-binding proteins (G-proteins) and modulates the activity of downstream effectors (PubMed:38632411). CASR is coupled with different G(q)/G(11), G(i)/G(o)- or G(s)-classes of G-proteins depending on the context (PubMed:38632411). In the parathyroid and kidney, CASR signals through G(q)/G(11) and G(i)/G(o) G-proteins: G(q)/G(11) coupling activates phospholipase C-beta, releasing diacylglycerol (DAG) and inositol 1,4,5-trisphosphate (IP3) second messengers, while G(i)/G(o) coupling mediates inhibition of adenylate cyclase activity (PubMed:38632411, PubMed:7759551). The G-protein- coupled receptor activity is activated by a co-agonist mechanism: aromatic amino acids, such as Trp or Phe, act concertedly with divalent cations, such as calcium or magnesium, to achieve full receptor activation (PubMed:27386547, PubMed:27434672, PubMed:32817431, PubMed:33603117, PubMed:34194040). Acts as an activator of the NLRP3 inflammasome via G(i)/G(o)-mediated signaling: down-regulation of cyclic AMP (cAMP) relieving NLRP3 inhibition by cAMP (PubMed:32843625). Acts as a regulator of proton-sensing receptor GPR68 in a seesaw manner: CASR-mediated signaling inhibits GPR68 signaling in response to extracellular calcium, while GPR68 inhibits CASR in presence of extracellular protons (By similarity).

Cellular Location
Cell membrane; Multi-pass membrane protein

Tissue Location
Expressed in the temporal lobe, frontal lobe, parietal lobe, hippocampus, and cerebellum. Also found in kidney, lung, liver, heart, skeletal muscle, placenta.

CASR Blocking Peptide (C-term) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

CASR Blocking Peptide (C-term) - Images

CASR Blocking Peptide (C-term) - Background

Senses changes in the extracellular concentration of calcium ions. The activity of this receptor is mediated by a G- protein that activates a phosphatidylinositol-calcium second messenger system.

CASR Blocking Peptide (C-term) - References

Pearce S.H.S.,et al.Submitted (DEC-1994) to the EMBL/GenBank/DDBJ databases.

Garrett J.E.,et al.J. Biol. Chem. 270:12919-12925(1995).
Aida K.,et al.Biochem. Biophys. Res. Commun. 214:524-529(1995).
Freichel M.,et al.Endocrinology 137:3842-3848(1996).
Aida K.,et al.J. Clin. Endocrinol. Metab. 80:2594-2598(1995).