abcam

Product datasheet

KCNJ1 peptide ab86648

Overview

Product name KCNJ1 peptide

Description

Nature Synthetic

Specifications

Our Abpromise guarantee covers the use of **ab86648** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications Blocking - Blocking peptide for Anti-KCNJ1 antibody (ab75906)

Purity 70 - 90% by HPLC.

Form Liquid

Additional notes

- First try to dissolve a small amount of peptide in either water or buffer. The more charged residues on a peptide, the more soluble it is in aqueous solutions.
- If the peptide doesn't dissolve try an organic solvent e.g. DMSO, then dilute using water or buffer.
- Consider that any solvent used must be compatible with your assay. If a peptide does not dissolve and you need to recover it, lyophilise to remove the solvent.
- Gentle warming and sonication can effectively aid peptide solubilisation. If the solution is cloudy or has gelled the peptide may be in suspension rather than solubilised.
- Peptides containing cysteine are easily oxidised, so should be prepared in solution just prior to use.

Preparation and Storage

Stability and Storage Shipped at 4°C. Upon delivery aliquot and store at -20°C or -80°C. Avoid repeated freeze / thaw cycles.

Information available upon request.

General Info

Function	In the kidney, probably plays a major role in potassium homeostasis. Inward rectifier potassium channels are characterized by a greater tendency to allow potassium to flow into the cell rather than out of it. Their voltage dependence is regulated by the concentration of extracellular potassium; as external potassium is raised, the voltage range of the channel opening shifts to more positive voltages. The inward rectification is mainly due to the blockage of outward current by internal magnesium. This channel is activated by internal ATP and can be blocked by external barium.
Tissue specificity	In the kidney and pancreatic islets. Lower levels in skeletal muscle, pancreas, spleen, brain, heart and liver.
Involvement in disease	Defects in KCNJ1 are the cause of Bartter syndrome type 2 (BS2) [MIM:241200]; also termed hyperprostanglandin E syndrome 2. BS refers to a group of autosomal recessive disorders characterized by impaired salt reabsorption in the thick ascending loop of Henle with pronounced salt wasting, hypokalemic metabolic alkalosis, and varying degrees of hypercalciuria. BS2 is a life-threatening condition beginning in utero, with marked fetal polyuria that leads to polyhydramnios and premature delivery. Another hallmark of BS2 is a marked hypercalciuria and, as a secondary consequence, the development of nephrocalcinosis and osteopenia.
Sequence similarities	Belongs to the inward rectifier-type potassium channel (TC 1.A.2.1) family. KCNJ1 subfamily.
Cellular localization	Membrane.

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