

KCNN2 Antibody (Internal)
Goat Polyclonal Antibody
Catalog # ALS13387**Specification**

KCNN2 Antibody (Internal) - Product Information

Application	WB, IHC-P, E
Primary Accession	Q9H2S1
Reactivity	Human, Monkey
Host	Goat
Clonality	Polyclonal
Calculated MW	64kDa KDa
Dilution	WB~~1:1000 IHC-P~~N/A E~~N/A

KCNN2 Antibody (Internal) - Additional Information**Gene ID** 3781**Other Names**

Small conductance calcium-activated potassium channel protein 2, SK2, SKCa 2, SKCa2, KCa2.2, KCNN2

Target/Specificity

Human KCNN2. This antibody is expected to recognize both isoforms (NP_067627.2; NP_740721.1).

Reconstitution & Storage

Store at -20°C. Minimize freezing and thawing.

Precautions

KCNN2 Antibody (Internal) is for research use only and not for use in diagnostic or therapeutic procedures.

KCNN2 Antibody (Internal) - Protein Information**Name** KCNN2 ([HGNC:6291](#))**Function**

Small conductance calcium-activated potassium channel that mediates the voltage-independent transmembrane transfer of potassium across the cell membrane through a constitutive interaction with calmodulin which binds the intracellular calcium allowing its opening (PubMed:10991935, PubMed:33242881, PubMed:9287325). The current is characterized by a voltage-independent activation, an intracellular calcium concentration increase-dependent activation and a single- channel conductance of about 3 picosiemens

(PubMed:10991935). Also presents an inwardly rectifying current, thus reducing its already small outward conductance of potassium ions, which is particularly the case when the membrane potential displays positive values, above + 20 mV (PubMed:10991935). The inward rectification could be due to a blockade of the outward current by intracellular divalent cations such as calcium and magnesium and could also be due to an intrinsic property of the channel pore, independent of intracellular divalent ions. There are three positively charged amino acids in the S6 transmembrane domain, close to the pore, that collectively control the conductance and rectification through an electrostatic mechanism. Additionally, electrostatic contributions from these residues also play an important role in determining the intrinsic open probability of the channel in the absence of calcium, affecting the apparent calcium affinity for activation. Forms an heteromeric complex with calmodulin, which is constitutively associated in a calcium-independent manner. Channel opening is triggered when calcium binds the calmodulin resulting in a rotary movement leading to the formation of the dimeric complex to open the gate (By similarity). Plays a role in the repolarization phase of cardiac action potential (PubMed:13679367).

Cellular Location

Membrane; Multi-pass membrane protein. Cytoplasm, myofibril, sarcomere, Z line {ECO:0000250|UniProtKB:P58390}

Tissue Location

Expressed in atrial myocytes (at protein level) (PubMed:13679367). Widely expressed.

KCNN2 Antibody (Internal) - Protocols

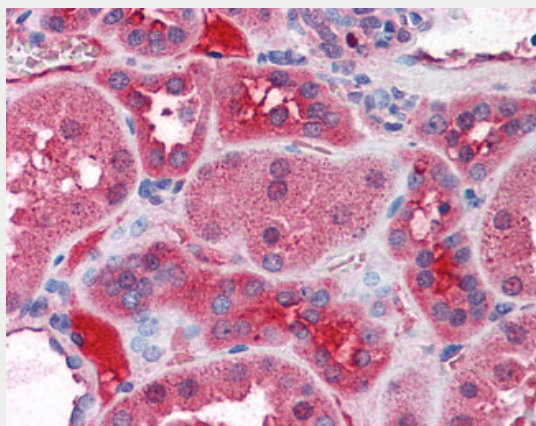
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

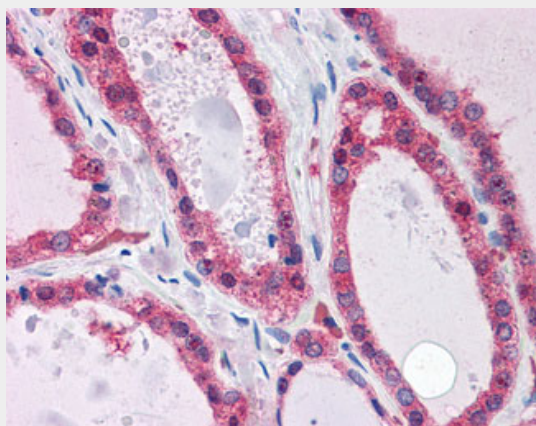
KCNN2 Antibody (Internal) - Images



KCNN2 antibody (0.3 ug/ml) staining of Human Liver lysate (35 ug protein in RIPA buffer).



Anti-KCNN2 antibody IHC of human kidney.



Anti-KCNN2 antibody IHC of human thyroid.

KCNN2 Antibody (Internal) - Background

Forms a voltage-independent potassium channel activated by intracellular calcium. Activation is followed by membrane hyperpolarization. Thought to regulate neuronal excitability by contributing to the slow component of synaptic afterhyperpolarization. The channel is blocked by apamin.

KCNN2 Antibody (Internal) - References

- Desai R.,et al.J. Biol. Chem. 275:39954-39963(2000).
Xu Y.,et al.J. Biol. Chem. 278:49085-49094(2003).
Mazzone J.N.,et al.Submitted (JUL-2001) to the EMBL/GenBank/DDBJ databases.
Ota T.,et al.Nat. Genet. 36:40-45(2004).
Schmutz J.,et al.Nature 431:268-274(2004).