

KIR3.4 (H-60): sc-50411

BACKGROUND

G protein-coupled inwardly rectifying potassium channels (KIR3.1 through KIR3.4) are coupled to numerous neurotransmitter receptors in the brain and are abundantly expressed in the olfactory bulb, hippocampus, neocortex, dentate gyrus, cerebellar cortex and thalamus regions of the brain. Also known as GIRK, KIR3 potassium channels localize to the soma and dendrites as well as axons of neurons. Liberated $G_{\beta\gamma}$ subunits from G protein heterotrimers bind to and regulate KIR3 channel activity. $G_{\beta 3}$ - and $G_{\beta 4}$ -containing $G_{\beta\gamma}$ dimers bind directly to cytoplasmic domains of KIR3 proteins and increase the K^+ current while $G_{\beta 5}$ -containing $G_{\beta\gamma}$ dimers inhibit KIR3 K^+ current. KIR3 activity is also inhibited by tyrosine phosphorylation. Brain-derived neurotrophic factor activates receptor tyrosine kinase-B, which then phosphorylates KIR3 tyrosine residues, effectively inactivating the KIR3 channels.

REFERENCES

- Braun, A.P., et al. 1992. Activation of α 1-adrenoceptors modulates the inwardly rectifying potassium currents of mammalian atrial myocytes. *Pflugers Arch.* 421: 431-439.
- Ponce, A., et al. 1996. G protein-gated inward rectifier K^+ channel proteins (GIRK1) are present in the soma and dendrites as well as in nerve terminals of specific neurons in the brain. *J. Neurosci.* 16: 1990-2001.
- Farkas, R.H., et al. 1997. Neurotensin and dopamine D2 activation oppositely regulate the same K^+ conductance in rat midbrain dopaminergic neurons. *Neurosci. Lett.* 231: 21-24.
- Lei, Q., et al. 2000. Activation and inhibition of G protein-coupled inwardly rectifying potassium (KIR3) channels by G protein by subunits. *Proc. Natl. Acad. Sci. USA* 97: 9771-9776.
- Rogalski, S.L., et al. 2000. TrkB activation by brain-derived neurotrophic factor inhibits the G protein-gated inward rectifier KIR3 by tyrosine phosphorylation of the channel. *J. Biol. Chem.* 275: 25082-25088.

CHROMOSOMAL LOCATION

Genetic locus: KCNJ5 (human) mapping to 11q24.3; Kcnj5 (mouse) mapping to 9 A4.

SOURCE

KIR3.4 (H-60) is a rabbit polyclonal antibody raised against amino acids 1-60 mapping within an N-terminal cytoplasmic domain of KIR3.4 of human origin.

PRODUCT

Each vial contains 200 μ g IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

APPLICATIONS

KIR3.4 (H-60) is recommended for detection of KIR3.4 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

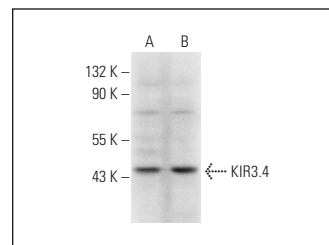
KIR3.4 (H-60) is also recommended for detection of KIR3.4 in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for KIR3.4 siRNA (h): sc-42622, KIR3.4 siRNA (m): sc-42623, KIR3.4 shRNA Plasmid (h): sc-42622-SH, KIR3.4 shRNA Plasmid (m): sc-42623-SH, KIR3.4 shRNA (h) Lentiviral Particles: sc-42622-V and KIR3.4 shRNA (m) Lentiviral Particles: sc-42623-V.

Molecular Weight of KIR3.4: 48 kDa.

Positive Controls: mouse heart extract: sc-2254, HeLa whole cell lysate: sc-2200 or Caki-1 cell lysate: sc-2224.

DATA



KIR3.4 (H-60): sc-50411. Western blot analysis of KIR3.4 expression in HeLa (A) and Caki-1 (B) whole cell lysates.

SELECT PRODUCT CITATIONS

- Wu, L., et al. 2012. Low-level ganglionated plexus stimulation facilitates atrial fibrillation: *in vivo* and *in vitro* studies. *Auton. Neurosci.* 168: 58-65.

PROTOCOLS

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