SANTA CRUZ BIOTECHNOLOGY, INC.

KCNQ2 (N-19): sc-7793

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Epilepsy affects about 0.5% of the world's population and has a large genetic component. Epilepsy results from an electrical hyperexcitability in the central nervous system. Potassium channels are important regulators of electrical signaling, determining the firing properties and responsiveness of a variety of neurons. Benign familial neonatal convulsions (BFNC), an autosomal dominant epilepsy of infancy, has been shown to be caused by mutations in the KCNQ2 or the KCNQ3 potassium channel genes. KCNQ2 and KCNQ3 are voltage-gated potassium channel proteins with six putative transmembrane domains. Both proteins display a broad distribution within the brain, with expression patterns that largely overlap.

REFERENCES

1. Singh, N.A., et al. 1998. A novel potassium channel gene, KCNQ2, is mutated in an inherited epilepsy of newborns. Nat. Genet. 18: 25-29.
2. Schroeder, B.C., et al. 1998. Moderate loss of function of cyclic-AMP-modulated KCNQ2/KCNQ3 K^+ channels causes epilepsy. Nature 396: 687-690.

CHROMOSOMAL LOCATION

Genetic locus: KCNQ2 (human) mapping to 20q13.33; Kcnq2 (mouse) mapping to 2 H4.

SOURCE

KCNQ2 (N-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of KCNQ2 of human origin.

PRODUCT

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

Blocking peptide available for competition studies, sc-7793 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

KCNQ2 (N-19) is recommended for detection of KCNQ2 and KQT2 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg 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).

KCNQ2 (N-19) is also recommended for detection of KCNQ2 and KQT2 in additional species, including avian.

Molecular Weight of KCNQ2: 120 kDa.

Positive Controls: rat cerebellum extract: sc-2398, rat brain extract: sc-2392 or KCNQ2 (h): 293T Lysate: sc-128912.

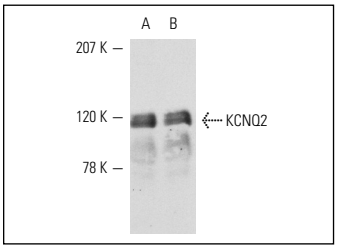
STORAGE

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

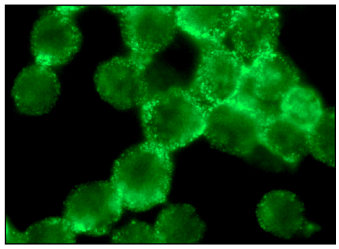
RESEARCH USE

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

DATA



KCNQ2 (N-19): sc-7793. Western blot analysis of KCNQ2 expression in rat cerebellum (**A**) and rat whole brain (**B**) extracts.



KCNQ2 (N-19): sc-7793. Immunofluorescence staining of methanol-fixed EOC 20 cells showing membrane localization.

SELECT PRODUCT CITATIONS

1. Martire, M., et al. 2004. M channels containing KCNQ2 subunits modulate norepinephrine, aspartate, and GABA release from hippocampal nerve terminals. J. Neurosci. 24: 592-597.
2. Soldovier, M.V., et al. 2006. Decreased subunit stability as a novel mechanism for potassium current impairment by a KCNQ2 C terminus mutation causing benign familial neonatal convulsions. J. Biol. Chem. 281: 418-428.
3. Schwake, M., et al. 2006. Structural determinants of M-type KCNQ (Kv7) K^+ channel assembly. J. Neurosci. 26: 3757-3766.
4. Martire, M., et al. 2007. Involvement of KCNQ2 subunits in [^{3}H]Dopamine release triggered by depolarization and pre-synaptic muscarinic receptor activation from rat striatal synaptosomes. J. Neurochem. 102: 179-193.
5. Luisi, R., et al. 2009. Activation of pre-synaptic M-type K^+ channels inhibits [^{3}H]D-aspartate release by reducing Ca^{2+} entry through P/Q-type voltage-gated Ca^{2+} channels. J. Neurochem. 109: 168-181.
6. Regev, N., et al. 2009. Selective interaction of syntaxin 1A with KCNQ2: possible implications for specific modulation of presynaptic activity. PLoS ONE 4: e6586.
7. Ishii, A., et al. 2009. A *de novo* KCNQ2 mutation detected in non-familial benign neonatal convulsions. Brain Dev. 31: 27-33.
8. Han, S.S., et al. 2010. NFκB/STAT3/PI3K signaling crosstalk in iMyc E µ B lymphoma. Mol. Cancer 9: 97.
9. Roza, C., et al. 2011. Accumulation of Kv7.2 channels in putative ectopic transduction zones of mice nerve-end neuromas. Mol. Pain 7: 58.