**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**


**SOURCE**

KCNQ3 (N-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the N-terminus of KCNQ3 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-7794 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

**STORAGE**

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

**PROTOCOLS**

See our web site at www.scbt.com or our catalog for detailed protocols and support products.