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ProductInformation

Anti-Potassium Channel GIRK2
Developed in Rabbit
Affinity Isolated Antibody

Product Number **P 2485**

Product Description

Anti-Potassium Channel GIRK2 (G-protein-activated inwardly rectifying potassium channel, Kir3.2) is developed in rabbit using a highly purified peptide CEELTERNGDVANLENESK, corresponding to amino acid residues 407-424 of mouse GIRK2[1] with additional N-terminal cysteine as the immunogen. The antibody was affinity isolated on immobilized immunogen.

Anti-Potassium Channel GIRK2 specifically recognizes GIRK2 protein and may be used for the detection of GIRK2 protein (approx. 43 kDa) in total brain extracts from mouse or rat by immunoblotting and immunohistochemistry.

Potassium channels contribute to maintaining cell volume, membrane potential, neuronal excitability and the secretion of transmitters, salt and hormones. Two families of potassium channels have been identified. One family includes the inwardly rectifying potassium channels, whereas the other family includes: voltage-sensing (KV); big conductance, calcium activated (BK_{CA}); and small conductance, calcium activated (SK) potassium channels. [1,2]

The G-protein-activated inwardly rectifying potassium channels (GIRKs) are part of a superfamily of inwardly rectifying potassium channels which include seven members. Four GIRKs, also referred to as Kir3.1-3.4, have been identified in mammals.[3] A fifth GIRK has been indentified in Xenopus oocytes. GIRK1-3 are widely expressed in the brain, where they are activated by at least eight neurotransmitters. GIRK4 has a limited distribution in the brain. In addition to G-proteins, GIRKs are also modulated by phosphatidylinositol 4,5-bisphosphate, intracellular sodium, ethanol and mechanical stretch.

Native channels are tetrameric and may be composed of several combinations of GIRK subunits.[4] GIRK1 cannot form functional channels by itself and GIRK1/2 is the predominant heterotetramer in brain although it is possible to isolate functional GIRK2/3 channels in brain. Regardless of subunit composition, GIRK channels function to dampen neuronal excitability. The functional significance of subunit composition is poorly understood. GIRK1/4 form functional channels in the atrial and sinoatrial node cells of the heart where they contribute to the regulation of cardiac rate.

Reagent

Anti-Potassium Channel GIRK2 is supplied as 25 µg of affinity isolated antibody at approximately 50 µg/ml in 0.05 M sodium phosphate buffer containing 0.2 % gelatin and 0.1 % sodium azide.

Precautions and Disclaimer

Due to the sodium azide content, a material safety data sheet (MSDS) for this product has been sent to the attention of the safety officer of your institution. Consult the MSDS for information regarding hazardous and safe handling practices.

Storage/Stability

Store the antibody at 2-8 °C. **Do not freeze the antibody.** For extended storage, freeze in working aliquots. Repeated freezing and thawing is not recommended. Storage in “frost-free” freezers is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours.

Product Profile

The recommended working dilution is 2 µg/ml for immunoblotting of total brain extracts using peroxidase conjugated-goat anti-rabbit IgG and detection by chemiluminescence. In addition to the 43 kDa band, a non-specific higher molecular weight band has also been observed at 45 kDa.

The recommended working dilution for immuno-histochemistry on frozen floating mouse brain sections fixed in 4% paraformaldehyde is 1.5-2.5 µg/ml with detection by DAB (diaminobenzidine).

Note: In order to obtain the best results and assay sensitivities of various techniques and preparations, we recommend determining optimal working dilutions by titration.

References

1. McFarlane, S. and Pollock, N.S., J. Neurosci., **20**, 1020 (2000).
2. Teschemacher, A.G. et al., Br. J. Pharmacol., **128**, 479 (1999).
3. Mark, M.D. and Herlitze, S., Eur. J. Biochem., **267**, 5830 (2000).
4. Jelacic, T.M. et al., J. Biol. Chem., **275**, 36211 (2000).

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