

Na⁺ CP type V α (4G8:1G7): sc-81631

BACKGROUND

Voltage-gated sodium channels drive the initial depolarization phase of the cardiac action potential and, therefore, critically determine conduction of excitation through the heart. The sodium channel gene SCN5A, which encodes the Na⁺ CP type V α protein, possesses two fundamental properties, ion conduction and gating. The human SCN5A gene maps to chromosome 3q21-24. Deletions or loss-of-function mutations in SCN5A result in a wide range of arrhythmias, including bradycardia, atrioventricular conduction delay and ventricular fibrillation. Specifically, patients with Brugada syndrome have mutations in the SCN5A gene, which reduces the sodium current. Additionally, gain-of-function mutations are associated with long QT syndrome type III (LQT3), a cardiac disorder that causes sudden death from ventricular tachyarrhythmias, specifically torsade de pointes. The SCN5A gene is expressed in human atrial and ventricular cardiac muscle, but not in adult skeletal muscle, brain, myometrium, liver or spleen.

REFERENCES

1. Wang, Q., Bowles, N.E. and Towbin, J.A. 1998. The molecular basis of long QT syndrome and prospects for therapy. *Mol. Med. Today* 4: 382-388.
2. Wang, Q., Chen, Q. and Towbin, J.A. 1998. Genetics, molecular mechanisms and management of long QT syndrome. *Ann. Med.* 30: 58-65.
3. Cerrone, M., Crotti, L., Faggiano, G., De Michelis, V., Napolitano, C., Schwartz, P.J. and Priori, S.G. 2001. Long QT syndrome and Brugada syndrome: two aspects of the same disease? *Ital. Heart J.* 2: 253-257.
4. Grant, A.O. 2001. Molecular biology of sodium channels and their role in cardiac arrhythmias. *Am. J. Med.* 110: 296-305.
5. Clancy, C.E. and Rudy, Y. 2002. Na⁺ channel mutation that causes both Brugada and long-QT syndrome phenotypes: a simulation study of mechanism. *Circulation* 105: 1208-1213.
6. Papadatos, G.A., Wallerstein, P.M., Head, C.E., Ratcliff, R., Brady, P.A., Benndorf, K., Saumarez, R.C., Trezise, A.E., Huang, C.L., Vandenberg, J.I., Colledge, W.H. and Grace, A.A. 2002. Slowed conduction and ventricular tachycardia after targeted disruption of the cardiac sodium channel gene SCN5A. *Proc. Natl. Acad. Sci. USA* 99: 6210-6215.

CHROMOSOMAL LOCATION

Genetic locus: SCN5A (human) mapping to 3p22.2.

SOURCE

Na⁺ CP type V α (4G8:1G7) is a mouse monoclonal antibody raised against a synthetic peptide corresponding to an extracellular region of Na⁺ CP type V α of human origin.

PRODUCT

Each vial contains 200 μ g IgG₁ in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

RESEARCH USE

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

APPLICATIONS

Na⁺ CP type V α (4G8:1G7) is recommended for detection of Voltage-gated sodium channel subunit α Nav1.5 of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000) and immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500).

Suitable for use as control antibody for Na⁺ CP type V α siRNA (h): sc-42640, Na⁺ CP type V α shRNA Plasmid (h): sc-42640-SH and Na⁺ CP type V α shRNA (h) Lentiviral Particles: sc-42640-V.

Molecular Weight of Na⁺ CP type V α : 260 kDa.

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 for detailed protocols and support products.