

Na Channel β1 Subunit (D4Z2N) Rabbit



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Applications: W, IP	Reactivity: H M R	Sensitivity: Endogenous	MW (kDa): 38	Source/Isotype: Rabbit IgG	UniProt ID: #Q07699	Entrez-Gene Id: 6324
Product Usage Information		Application Western Blotting Immunoprecipitation		Dilution 1:1000 1:50		
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 μg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.				
Specificity/Sensitivity		Na Channel β 1 Subunit (D4Z2N) Rabbit mAb recognizes endogenous levels of total sodium channel β 1 subunit protein.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues near the carboxy terminus of human sodium channel $\beta 1$ subunit protein.				
Background		Mammalian voltage-gated sodium channels (VGSCs) are composed of a pore-forming α subunit and one or more regulatory β subunits (1). Four separate genes (SCN1B-SCN4B) encode the five mammalian β subunits β 1, β 18, β 2, β 3, and β 4. In general, β subunit proteins are type I transmembrane proteins, with the exception of secreted β 1B protein (reviewed in 2). β subunits regulate α subunit gating and kinetics, which controls cell excitability (3,4). Sodium channel β subunits also function as Ig superfamily cell adhesion molecules that regulate cell adhesion and migration (5,6). Additional research reveals sequential processing of β subunit proteins by β -secretase (BACE1) and γ secretase, resulting in ectodomain shedding of β subunit and generation of an intracellular carboxy-terminal fragment (CTF). Generation of the CTF is thought to play a role in cell adhesion and migration (7,8). Multiple studies demonstrate a link between β subunit gene mutations and a number of disorders, including epilepsy, cardiac arrhythmia, multiple sclerosis, neuropsychiatric disorders, neuropathy, inflammatory pain, and cancer (9-13). The sodium channel β 1 subunit (SCN1B) plays a crucial role in neuronal migration and pathfinding during brain development (14). Mutations in the corresponding <i>SCN1B</i> gene are associated with generalized epilepsy with febrile seizures plus 1 (15), Brugada syndrome (16), and familial atrial fibrillation (17). A <i>SCN1B</i> loss of function mutation results in a severe form of pediatric epileptic encephalopathy known as Dravet syndrome (18).				
Background References		1. Catterall, W.A. (1992) <i>Physiol Rev</i> 72, S15-48. 2. Catterall, W.A. (2012) <i>J Physiol</i> 590, 2577-89. 3. Isom, L.L. et al. (1992) <i>Science</i> 256, 839-42. 4. Brackenbury, W.J. and Isom, L.L. (2011) <i>Front Pharmacol</i> 2, 53. 5. Isom, L.L. et al. (1995) <i>Cell</i> 83, 433-42. 6. Malhotra, J.D. et al. (2000) <i>J Biol Chem</i> 275, 11383-8. 7. Wong, H.K. et al. (2005) <i>J Biol Chem</i> 280, 23009-17. 8. Kim, D.Y. et al. (2005) <i>J Biol Chem</i> 280, 23251-61. 9. Wallace, R.H. et al. (1998) <i>Nat Genet</i> 19, 366-70. 10. Lopez-Santiago, L.F. et al. (2007) <i>J Mol Cell Cardiol</i> 43, 636-47. 11. Chioni, A.M. et al. (2009) <i>Int J Biochem Cell Biol</i> 41, 1216-27. 12. O'Malley, H.A. et al. (2009) <i>Mol Cell Neurosci</i> 40, 143-55. 13. Valdivia, C.R. et al. (2010) <i>Cardiovasc Res</i> 86, 392-400. 14. Brackenbury, W.J. et al. (2013) <i>Proc Natl Acad Sci U S A</i> 110, 1089-94. 15. Meadows, L.S. et al. (2002) <i>J Neurosci</i> 22, 10699-709. 16. Hu, D. et al. (2012) <i>Heart Rhythm</i> 9, 760-9. 17. Li, R.G. et al. (2013) <i>Int J Mol Med</i> 32, 144-50. 18. Patino, G.A. et al. (2009) <i>J Neurosci</i> 29, 10764-78.				

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4° C with gentle shaking, overnight.

Applications Key W: Western Blotting IP: Immunoprecipitation

Cross-Reactivity Key H: Human M: Mouse R: Rat

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