

hERG1a (D1Y2J) Rabbit mAb



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Applications: W	Reactivity: H R	Sensitivity: Endogenous	MW (kDa): 135, 155	Source/Isotype: Rabbit IgG	UniProt ID: #Q12809	Entrez-Gene Id: 3757
Product Usage	1	Application			Dilution	
Information	•	Western Blotting			1:1000	
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		hERG1a (D1Y2J) Rabbit mAb recognizes endogenous levels of both mature and immature hERG1a protein. This antibody cross-reacts with proteins of unknown origin at 65 and 42 kDa in some cell lines. This antibody does not recognize hERG1b protein.				
Species predicted to react based on 100% sequence homology		Monkey				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Ala223 of human hERG1a protein.				
Background		hERG1(human ether-a-go-go-related gene potassium channel 1) is a voltage gated potassium channel alpha-subunit which mediates the rapidly activating component of the delayed rectifying potassium				

current in heart (IKr) (1,2). The hERG channel is composed of two subunits, 1a and 1b, which differ at amino terminus due to alternative splicing. Native hERG channels are heteromers of hERG1a with hERG1b. Both subunits contribute to IKr current (3-6).

Blockade of hERG currents induced by compounds or mutation of hERG encoding gene-KCNH2 causes

Blockade of hERG currents induced by compounds or mutation of hERG encoding gene-KCNH2 causes ventricular arrhythmias associated with inherited and acquired long QT syndrome and cardiomyocyte apoptosis (7-10). Therefore, hERG channel is a primary target for the development of class III antiarrhythmic agents (11,12). The hERG channel is also inhibited by a variety of non-antiarrhythmic compounds, which result in side effects. Consequently, hERG channel blockage is a common counter screen when selecting therapeutic agents for various diseases (11,13,14).

Research studies have implicated hERG in cancer cell survival (15). In normal human adult tissue, hERG is expressed in heart, brain, myometrium, pancreas, and hematopoietic progenitors (16,17). hERG is expressed in various cancer cell lines of epithelial, neuronal, leukemic, and connective tissue origin but not in corresponding normal cells (18-22). Furthermore, hERG expression is associated with enhanced cancer cell proliferation, invasiveness, and poor prognosis (23,24).

Background References

- 1. Warmke, J.W. and Ganetzky, B. (1994) Proc Natl Acad Sci U S A 91, 3438-42.
- 2. Sanguinetti, M.C. and Tristani-Firouzi, M. (2006) Nature 440, 463-9.
- 3. Lees-Miller, J.P. et al. (1997) Circ Res 81, 719-26.
- 4. London, B. et al. (1997) Circ Res 81, 870-8.
- 5. Lees-Miller, J.P. et al. (2003) Mol Cell Biol 23, 1856-62.
- 6. Sale, H. et al. (2008) *Circ Res* 103, e81-95.
- 7. Curran, M.E. et al. (1995) *Cell* 80, 795-803.
- 8. Itoh, T. et al. (1998) Hum Genet 102, 435-9.
- 9. González-Juanatey, J.R. et al. (2003) Circulation 107, 127-31.
- 10. Gong, Q. et al. (2006) J Biol Chem 281, 4069-74.
- 11. Thomas, D. et al. (2006) Curr Pharm Des 12, 2271-83.
- 12. Staudacher, I. et al. (2010) Curr Opin Drug Discov Devel 13, 23-30.
- 13. Wible, B.A. et al. (2005) *J Pharmacol Toxicol Methods* 52, 136-45.
- 14. Yang, B.F. et al. (2004) Acta Pharmacol Sin 25, 554-60.
- 15. Jehle, J. et al. (2011) Cell Death Dis 2, e193.
- 16. Pond, A.L. et al. (2000) / Biol Chem 275, 5997-6006.
- 17. Rosati, B. et al. (2000) *FASEB J* 14, 2601-10.
- 18. Smith, G.A. et al. (2002) J Biol Chem 277, 18528-34.
- 19. Lastraioli, E. et al. (2004) *Cancer Res* 64, 606-11.

20. Masi, A. et al. (2005) Br J Cancer 93, 781-92. 21. Lin, H. et al. (2007) / Cell Physiol 212, 137-47. 22. Gong, J.H. et al. (2010) Oncol Rep 23, 1747-56.

23. Ding, X.W. et al. (2008) J Surg Oncol 97, 57-62. 24. Shao, X.D. et al. (2008) Cancer Biol Ther 7, 45-50.

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

Cross-Reactivity Key

H: Human R: Rat

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