Revision 1

Store at -20C

#14562

Phospholamban (D9W8M) Rabbit mAb

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W	H M R	Endogenous	12, 24	Rabbit IgG	#P26678	5350

Product Usage Information

Application	Dilution
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

Phospholamban (D9W8M) Rabbit mAb recognizes endogenous levels of total phospholamban protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues near the amino terminus of human phospholamban protein.

Background

Phospholamban (PLN) was identified as a major phosphoprotein component of the sarcoplasmic reticulum (SR) (1). Its name, "lamban", is derived from the greek word "lambano" meaning "to receive", so named due to the fact that phospholamban is heavily phosphorylated on serine and threonine residues in response to cardiac stimulation (1). Although originally thought to be a single 20-25 kDa protein due to its electrophoretic mobility on SDS-PAGE, PLN is actually a 52 amino acid, 6 kDa, membrane-spanning protein capable of forming stable homooligomers, even in the presence of SDS (2). Despite very high expression in cardiac tissue, phospholamban is also expressed in skeletal and smooth muscle (3). Localization of PLN is limited to the SR, where it serves as a regulator of the sarco-endoplasmic reticulum calcium ATPase, SERCA (4). PLN binds directly to SERCA and effectively lowers its affinity for calcium, thus reducing calcium transport into the SR. Phosphorylation of PLN at Ser16 by Protein Kinase A or myotonic dystrophy protein kinase and/or phosphorylation at Thr17 by Ca^{2+}/calmodulin-dependent protein kinase results in release of PLN from SERCA, relief of this inhibition, and increased calcium uptake by the SR (reviewed in 5,6). It has long been held that phosphorylation at Ser16 and Thr17 occurs sequentially, but increasing evidence suggests that phosphorylation, especially at Thr17, may be differentially regulated (reviewed in 7,8).

Rodent models of heart failure have shown that the expression level and degree of phosphorylation of PLN are critical in modulating calcium flux and contractility (reviewed in 9-11). Deletion or decreased expression of PLN promotes increased calcium flux and increased cardiac contractility, whereas overexpression of PLN results in sequestration of SERCA, decreased calcium flux, reduced contractility, and rescue of cardiac dysfunction and failure in mouse models of hypertension and cardiomyopathy (reviewed in 10). Distinct mutations in PLN have been detected in humans, resulting either in decreased or no expression of PLN protein (12,13) or binding defects between PLN, SERCA and/or regulatory proteins (14,15), both of which result in cardiac myopathy and heart failure. Interestingly, while the human phenotype of most PLN defects mimic those seen in rodent and vice versa, there are some instances where the type and severity of cardiac disease resulting from PLN mutations in rodent and human differ, making a consensus mechanism elusive.

Background References

1. Kirchberber, M.A. et al. (1975) *Recent Adv Stud Cardiac Struct Metab* 5, 103-15.
2. Zhan, Q.Q. et al. (1991) *J Biol Chem* 266, 21810-4.
3. Fujii, J. et al. (1991) *J Biol Chem* 266, 11669-75.
4. Tada, M. and Kirchberger, M.A. *Recent Adv Stud Cardiac Struct Metab* 11, 265-72.
5. Traaseth, N.J. et al. (2008) *Biochemistry* 47, 3-13.
6. Bhupathy, P. et al. (2007) *J Mol Cell Cardiol* 42, 903-11.
7. Hagemann, D. and Xiao, R.P. (2002) *Trends Cardiovasc Med* 12, 51-6.
8. Mattiazzi, A. et al. (2005) *Cardiovasc Res* 68, 366-75.
9. Chu, G. and Kranias, E.G. (2006) *Novartis Found Symp* 274, 156-71; discussion 172-5, 272-6.
10. Schwinger, R.H. and Frank, K.F. (2003) *Sci STKE* 2003, pe15.
11. MacLennan, D.H. and Kranias, E.G. (2003) *Nat Rev Mol Cell Biol* 4, 566-77.
12. Haghighi, K. et al. (2008) *Hum Mutat* 29, 640-7.
13. Haghighi, K. et al. (2003) *J Clin Invest* 111, 869-76.
14. Schmitt, J.P. et al. (2003) *Science* 299, 1410-3.
15. Haghighi, K. et al. (2006) *Proc Natl Acad Sci USA* 103, 1388-93.

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 **M:** Mouse **R:** Rat

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Limited Uses