

p-phospholamban (Thr 17)-R: sc-17024-R

BACKGROUND

Regulation of the heart contraction-relaxation cycle is controlled by the release and uptake of intracellular calcium by the Ca^{2+} -ATPase, SERCA2a. SERCA2a function is inhibited by a direct molecular interaction with phospholamban (also designated PLN and PLB), which is an integral membrane protein found primarily in the sarcoplasmic reticulum of cardiac muscle. After stimulation of β -adrenergic receptors, phospholamban is phosphorylated on either Ser 16 or Thr 17, which causes the release of SERCA2a. This release results in an increase in SERCA2a activity as well as an increase in the calcium concentration in the sarcoplasmic reticulum. SERCA2a activity is a major determinant of cardiac function, and therefore, phospholamban is thought to play a role in heart failure by mediating the level of calcium in the sarcoplasmic reticulum.

REFERENCES

1. Katz, A.M. 1998. Discovery of phospholamban. A personal history. *Ann. NY Acad. Sci.* 853: 9-19.
2. MacLennan, D.H., et al. 1998. Sites of regulatory interaction between calcium ATPases and phospholamban. *Ann. NY Acad. Sci.* 853: 31-42.
3. Colyer, J. 1998. Phosphorylation states of phospholamban. *Ann. NY Acad. Sci.* 853: 79-91.
4. Tada, M., et al. 1998. Molecular regulation of phospholamban function and gene expression. *Ann. NY Acad. Sci.* 853: 116-129.
5. Arai, M. 2000. Function and regulation of sarcoplasmic reticulum Ca^{2+} -ATPase: advances during the past decade and prospects for the coming decade. *Jpn. Heart J.* 41: 1-13.

CHROMOSOMAL LOCATION

Genetic locus: PLN (human) mapping to 6q22.31; Pln (mouse) mapping to 10 B3.

SOURCE

p-phospholamban (Thr 17)-R is a rabbit polyclonal antibody raised against a short amino acid sequence containing Thr 17 phosphorylated phospholamban of human origin.

PRODUCT

Each vial contains 200 μg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-17024 P, (100 μg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, ****DO NOT FREEZE****. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

RESEARCH USE

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

APPLICATIONS

p-phospholamban (Thr 17)-R is recommended for detection of Thr 17 phosphorylated phospholamban of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μg per 100-500 μg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

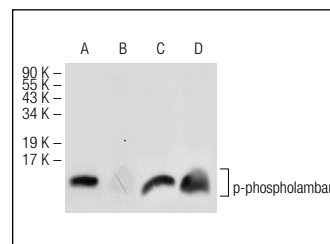
p-phospholamban (Thr 17)-R is also recommended for detection of correspondingly phosphorylated phospholamban in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for phospholamban siRNA (h): sc-39143, phospholamban siRNA (m): sc-39144, phospholamban shRNA Plasmid (h): sc-39143-SH, phospholamban shRNA Plasmid (m): sc-39144-SH, phospholamban shRNA (h) Lentiviral Particles: sc-39143-V and phospholamban shRNA (m) Lentiviral Particles: sc-39144-V.

Molecular Weight of p-phospholamban: 27 kDa.

Positive Controls: mouse heart extract: sc-2254.

DATA



Western blot analysis of phospholamban phosphorylation in untreated (A, C) and lambda protein phosphatase (sc-200312A) treated (B, D) mouse heart tissue extracts. Antibodies tested include p-phospholamban (Thr 17)-R: sc-17024-R (A, B) and phospholamban (L-15): sc-21923 (C, D).

SELECT PRODUCT CITATIONS

1. Benziane, B., et al. 2008. Divergent cell signaling after short-term intensified endurance training in human skeletal muscle. *Am. J. Physiol. Endocrinol. Metab.* 295: E1427-E1438.
2. Kushnir, A., et al. 2010. Role of CaMKII δ phosphorylation of the cardiac ryanodine receptor in the force frequency relationship and heart failure. *Proc. Natl. Acad. Sci. USA* 107: 10274-10279.
3. Moon, M.R., et al. 2012. Differential calcium handling in two canine models of right ventricular pressure overload. *J. Surg. Res.* 178: 554-562.
4. Kienesberger, P.C., et al. 2012. Myocardial ATGL overexpression decreases the reliance on fatty acid oxidation and protects against pressure overload-induced cardiac dysfunction. *Mol. Cell. Biol.* 32: 740-750.