SANTA CRUZ BIOTECHNOLOGY, INC.

p-PERK (Thr 981): sc-32577



BACKGROUND

PERK (eukaryotic translation initiation factor 2- α kinase 3, elF2 α kinase 3, PEK, WRS) is a Ser/Thr protein kinase, type I membrane protein of the endoplasmic reticulum that is abundant in secretory tissues and is inducible by ER-stress. PERK phosphorylates the α subunit of eukaryotic translation-initiation factor 2 (elF2), leading to its inactivation, a reduction of translation initiation and repression of global protein synthesis. PERK may serve as an effector of unfolded protein response (UPR)-induced G₁ growth arrest due to the loss of cyclin D1. PERK dimerizes with HSPA5/BIP in resting cells and oligomerizes in ER-stressed cells. Perturbation in protein folding in the endoplasmic reticulum (ER) can promote dissociation from HSPA5/BIP and oligomerization, resulting in autophosphorylation and kinase activity induction.

CHROMOSOMAL LOCATION

Genetic locus: EIF2AK3 (human) mapping to 2p11.2; Eif2ak3 (mouse) mapping to 6 C1.

SOURCE

p-PERK (Thr 981) is a rabbit polyclonal antibody raised against a short amino acid sequence containing Thr 981 phosphorylated PERK of human origin.

PRODUCT

Each vial contains 100 μg lgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-32577 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

p-PERK (Thr 981) is recommended for detection of Thr 981 phosphorylated PERK of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 μ g per 100-500 μ g 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-PERK (Thr 981) is also recommended for detection of correspondingly phosphorylated PERK in additional species, including equine, canine, bovine, porcine and avian.

Suitable for use as control antibody for PERK siRNA (h): sc-36213, PERK siRNA (m): sc-36214, PERK shRNA Plasmid (h): sc-36213-SH, PERK shRNA Plasmid (m): sc-36214-SH, PERK shRNA (h) Lentiviral Particles: sc-36213-V and PERK shRNA (m) Lentiviral Particles: sc-36214-V.

Molecular Weight of p-PERK: 125 kDa.

RESEARCH USE

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

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.

STORAGE

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

DATA



p-PERK (Thr 981): sc-32577. Western blot analysis of PERK phosphorylation in HeLa (**A**) and Thapsigargin treated HeLa (**B**) whole cell lysates.

SELECT PRODUCT CITATIONS

- Kubisch, C.H., et al. 2006. Early activation of endoplasmic reticulum stress is associated with arginine-induced acute pancreatitis. Am. J. Physiol. Gastrointest. Liver Physiol. 291: G238-G245.
- Fatma, N., et al. 2011. Deficiency of Prdx6 in lens epithelial cells induces ER stress response-mediated impaired homeostasis and apoptosis. Am. J. Physiol., Cell Physiol. 301: C954-C967.
- Xin, W., et al. 2011. Attenuation of endoplasmic reticulum stress-related myocardial apoptosis by SERCA2a gene delivery in ischemic heart disease. Mol. Med. 17: 201-210.
- Miranda, S., et al. 2012. Beneficial effects of fenofibrate in retinal pigment epithelium by the modulation of stress and survival signaling under diabetic conditions. J. Cell. Physiol. 227: 2352-2362.
- Liu, X.A., et al. 2012. Expression of the hyperphosphorylated tau attenuates ER stress-induced apoptosis with upregulation of unfolded protein response. Apoptosis 17: 1039-1049.
- Jiménez-Castro, M.B., et al. 2012. Tauroursodeoxycholic acid affects PPArg and TLR4 in steatotic liver transplantation. Am. J. Transplant. 12: 3257-3271.
- Fasano, E., et al. 2012. DHA induces apoptosis by altering the expression and cellular location of GRP78 in colon cancer cell lines. Biochim. Biophys. Acta 1822: 1762-1772.
- 8. Xu, M., et al. 2012. Activation of the unfolded protein response contributed to the selective cytotoxicity of oroxylin A in human hepatocellular carcinoma HepG2 cells. Toxicol. Lett. 212: 113-125.
- Maddalena, F., et al. 2013. Resistance to paclitxel in breast carcinoma cells requires a quality control of mitochondrial antiapoptotic proteins by TRAP1. Mol. Oncol. 7: 895-906.
- Jiang, Q., et al. 2013. ATF4 activation by the p38MAPK-elF4E axis mediates apoptosis and autophagy induced by selenite in Jurkat cells. FEBS Lett. 587: 2420-2429.