SANTA CRUZ BIOTECHNOLOGY, INC.

PERK (T-19): sc-9477

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

An interferon-inducible, RNA-dependent protein serine/threonine kinase (PKR) has been described. PKR in earlier literature is variously known as DAI, dsJ, PI kinase, p65, p67 or TIK for the mouse kinase; and p68 or p69 for the human kinase. The PKR kinase substrate is the α subunit of protein synthesis initiation factor eIF-2. Phosphorylation of eIF-2α on serine-51 results in inhibition of translation. The serine/threonine kinase catalytic domains map to the carboxy terminal half of the protein while the RNA-binding domains are located in the amino terminal region. PERK is a type I transmembrane protein located in the endoplasmic reticulum (ER) that contains a kinase domain similar to the kinase domain of PKR. PERK is activated in response to ER stress and phosphorylates eIF-2α, thus inhibiting the translation of mRNA.

REFERENCES

1. Hershey, J.W.B., et al. 1989. Protein phosphorylation controls translation rates. J. Biol. Chem. 264: 20823-20826.
2. Meurs, E., et al. 1990. Molecular cloning and characterization of the human double-stranded RNA-activated protein kinase induced by interferon. Cell 62: 379-390.
3. Thomis, D.C., et al. 1992. Mechanism of interferon action: cDNA structure expression, and regulation of the interferon-induced, RNA-dependent P1/eIF-2 α protein kinase from human cells. Virology 188: 33-46.

CHROMOSOMAL LOCATION

Genetic locus: Eif2ak3 (mouse) mapping to 6 C1.

SOURCE

PERK (T-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the C-terminus of PERK of mouse origin.

PRODUCT

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

Blocking peptide available for competition studies, sc-9477 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

PERK (T-19) is recommended for detection of PERK of mouse and rat 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).

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

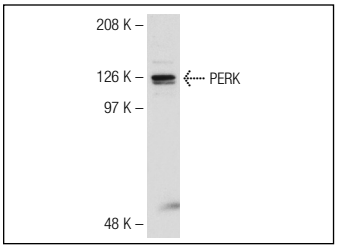
Molecular Weight of PERK: 125 kDa.

Positive Controls: NIH/3T3 whole cell lysate: sc-2210.

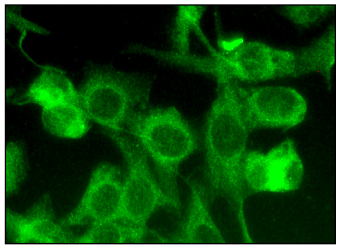
STORAGE

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

DATA



PERK (H-300): sc-9477. Western blot analysis of PERK expression in NIH/3T3 whole cell lysate.



PERK (T-19): sc-9477. Immunofluorescence staining of methanol-fixed NIH/3T3 cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Hayashi, T., et al. 2003. Induction of GRP 78 by ischemic preconditioning reduces endoplasmic reticulum stress and prevents delayed neuronal cell death. J. Cereb. Blood Flow Metab. 23: 949-961.
2. Hayashi, T., et al. 2003. Oxidative damage to the endoplasmic reticulum is implicated in ischemic neuronal cell death. J. Cereb. Blood Flow Metab. 23: 1117-1128.
3. Baltzis, D., et al. 2004. Resistance to vesicular stomatitis virus infection requires a functional cross talk between the eukaryotic translation initiation factor 2α kinases PERK and PKR. J. Virol. 78: 12747-12761.
4. Sokka, A.L., et al. 2007. Endoplasmic reticulum stress inhibition protects against excitotoxic neuronal injury in the rat brain. J. Neurosci. 27: 901-908.
5. Sanderson, T.H., et al. 2010. PKR-like endoplasmic reticulum kinase (PERK) activation following brain ischemia is independent of unfolded nascent proteins. Neuroscience 169: 1307-1314.
6. Samali, A., et al. 2010. Methods for monitoring endoplasmic reticulum stress and the unfolded protein response. Int. J. Cell Biol. 2010: 830307.
7. Caricilli, A.M., et al. 2011. Gut microbiota is a key modulator of Insulin resistance in TLR 2 knockout mice. PLoS Biol. 9: e1001212.
8. Lind, K.R., et al. 2013. The unfolded protein response to endoplasmic reticulum stress in cultured astrocytes and rat brain during experimental diabetes. Neurochem. Int. 62: 784-795.

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.