SANTA CRUZ BIOTECHNOLOGY, INC.

PERK (N-18): sc-9479

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.

CHROMOSOMAL LOCATION

Genetic locus: EIF2AK3 (human) mapping to 2p11.2.

SOURCE

PERK (N-18) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of PERK 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-9479 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

PERK (N-18) is recommended for detection of PERK of 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), immunohistochemistry (including paraffin-embedded sections) (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 (h): sc-36213, PERK shRNA Plasmid (h): sc-36213-SH and PERK shRNA (h) Lentiviral Particles: sc-36213-V.

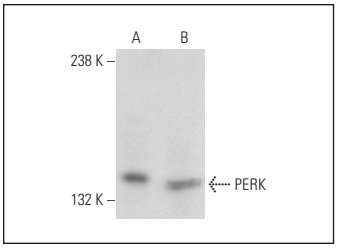
Molecular Weight of PERK: 125 kDa.

Positive Controls: K-562 whole cell lysate: sc-2203, Daudi cell lysate: sc-2415 or Hep G2 cell lysate: sc-2227.

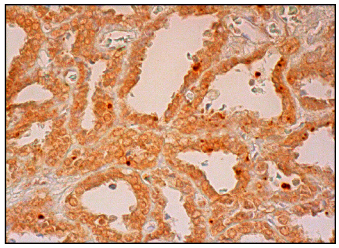
RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941. 4) Immunohistochemistry: use ImmunoCruz™: sc-2053 or ABC: sc-2023 goat IgG Staining Systems.

DATA



PERK (N-18): sc-9479. Western blot analysis of PERK expression in K-562 (**A**) and Daudi (**B**) whole cell lysates.



PERK (N-18): sc-9479. Immunoperoxidase staining of formalin fixed, paraffin-embedded human seminal vesicle tissue showing cytoplasmic and nuclear staining of glandular cells.

SELECT PRODUCT CITATIONS

1. Onuki, R., et al. 2004. An RNA-dependent protein kinase is involved in tunicamycin-induced apoptosis and Alzheimer's disease. EMBO J. 23: 959-968.
2. Chang, K.C., et al. 2011. Dominant expression of survival signals of endoplasmic reticulum stress response in Hodgkin lymphoma. Cancer Sci. 102: 275-281.
3. Liu, X.H., et al. 2011. Cardiomyocyte-specific disruption of Serca2 in adult mice causes sarco(endo)plasmic reticulum stress and apoptosis. Cell Calcium 49: 201-207.
4. Wu, X.D., et al. 2013. Hypoxic preconditioning protects microvascular endothelial cells against hypoxia/reoxygenation injury by attenuating endoplasmic reticulum stress. Apoptosis 18: 85-98.