SANTA CRUZ BIOTECHNOLOGY, INC.

PKR (D-20): sc-708

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Interferon-inducible RNA-dependent protein serine/threonine kinase, PKR, is variously designated in earlier literature 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. Molecular cDNA clones have been isolated from both human and mouse cells. 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. Three kinds of regulation of PKR enzymatic activity have been described. These include transcriptional regulation in response to interferon, an autoregulatory mechanism controlling PKR expression at the level of translation and post-translational regulation by RNA-mediated autophosphorylation.

CHROMOSOMAL LOCATION

Genetic locus: EIF2AK2 (human) mapping to 2p22.2; Eif2ak2 (mouse) mapping to 17 E3.

SOURCE

PKR (D-20) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of PKR of mouse 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-708 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

PKR (D-20) is recommended for detection of PKR of mouse, rat and, to a lesser extent, 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 PKR siRNA (h): sc-36263, PKR siRNA (m): sc-36264, PKR shRNA Plasmid (h): sc-36263-SH, PKR shRNA Plasmid (m): sc-36264-SH, PKR shRNA (h) Lentiviral Particles: sc-36263-V and PKR shRNA (m) Lentiviral Particles: sc-36264-V.

Molecular Weight of PKR: 68 kDa.

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

RESEARCH USE

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

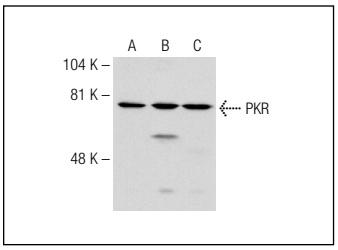
PROTOCOLS

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

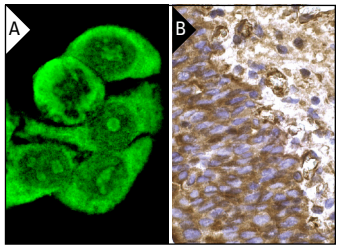
STORAGE

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

DATA



PKR (D-20): sc-708. Western blot analysis of PKR expression in A-431 (**A**), HeLa (**B**) and BJAB (**C**) whole cell lysates.



PKR (D-20): sc-708. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic staining (**A**). Immunoperoxidase staining of formalin fixed, paraffin-embedded human urinary bladder tissue showing cytoplasmic staining of urothelial cells (**B**).

SELECT PRODUCT CITATIONS

1. Iordanov, M.S., et al. 2001. Activation of NF-B by double-stranded RNA (dsRNA) in the absence of protein kinase R and RNase L demonstrates the existence of two separate dsRNA-triggered antiviral programs. Mol. Cell. Biol. 21: 61-72.
2. Gilfoy, F.D. and Mason, P.W. 2007. West Nile virus-induced interferon production is mediated by the double-stranded RNA-dependent protein kinase PKR. J. Virol. 81: 11148-11158.
3. Tseng, J.C., et al. 2007. Restricted tissue tropism and acquired resistance to Sindbis viral vector expression in the absence of innate and adaptive immunity. Gene Ther. 14: 1166-1174.
4. Oster, B., et al. 2008. Human herpesvirus 6B induces phosphorylation of p53 in its regulatory domain by a CK2- and p38-independent pathway. J. Gen. Virol. 89: 87-96.
5. Blalock, W.L., et al. 2009. PKR activity is required for acute leukemic cell maintenance and growth: a role for PKR-mediated phosphatase activity to regulate GSK-3 phosphorylation. J. Cell. Physiol. 221: 232-241.
6. Blalock, W.L., et al. 2011. Multiple forms of PKR present in the nuclei of acute leukemia cells represent an active kinase that is responsive to stress. Leukemia 25: 236-245.
7. Katta, A., et al. 2012. Diminished muscle growth in the obese Zucker rat following overload is associated with hyperphosphorylation of AMPK and dsRNA-dependent protein kinase. J. Appl. Physiol. 113: 377-384.