

PKR (B-10): sc-6282

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-2 α 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 (B-10) is a mouse monoclonal antibody raised against amino acids 1-515 of PKR of mouse origin.

PRODUCT

Each vial contains 200 μ g IgG_{2a} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

PKR (B-10) is available conjugated to agarose (sc-6282 AC), 500 μ g/0.25 ml agarose in 1 ml, for IP; to HRP (sc-6282 HRP), 200 μ g/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-6282 PE), fluorescein (sc-6282 FITC), Alexa Fluor[®] 488 (sc-6282 AF488), Alexa Fluor[®] 546 (sc-6282 AF546), Alexa Fluor[®] 594 (sc-6282 AF594) or Alexa Fluor[®] 647 (sc-6282 AF647), 200 μ g/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor[®] 680 (sc-6282 AF680) or Alexa Fluor[®] 790 (sc-6282 AF790), 200 μ g/ml, for Near-Infrared (NIR) WB, IF and FCM.

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APPLICATIONS

PKR (B-10) is recommended for detection of PKR 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 immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500).

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.

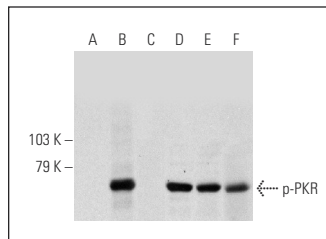
Molecular Weight of PKR: 68 kDa.

Positive Controls: NIH/3T3 whole cell lysate: sc-2210, A-431 whole cell lysate: sc-2201 or HeLa whole cell lysate: sc-2200.

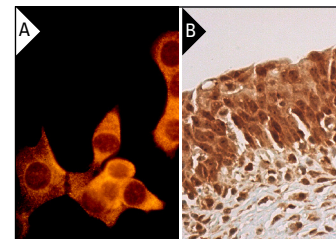
STORAGE

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

DATA



Western blot analysis of PKR phosphorylation in untreated (A,D), calyculin A treated (B,E) and lambda protein phosphatase (sc-200312A) treated (C,F) HeLa whole cell lysates. Antibodies tested include p-PKR (Thr 451): sc-101784 (A,B,C) and PKR (B-10): sc-6282 (D,E,F).



PKR (B-10): sc-6282. Immunofluorescence staining of methanol-fixed NIH/3T3 cells showing cytoplasmic localization (A). Immunoperoxidase staining of formalin fixed, paraffin-embedded human urinary bladder tissue showing nuclear and cytoplasmic staining of urothelial cells (B).

SELECT PRODUCT CITATIONS

- Farassati, F., et al. 2001. Oncogenes in Ras signalling pathway dictate host-cell permissiveness to herpes simplex virus 1. *Nat. Cell Biol.* 3: 745-750.
- Yim, H.C., et al. 2016. The kinase activity of PKR represses inflammasome activity. *Cell Res.* 26: 367-379.
- Ranganath, N., et al. 2016. Type I interferon responses are impaired in latently HIV infected cells. *Retrovirology* 13: 66.
- Zhang, M., et al. 2016. AIM2 inflammasome mediates arsenic-induced secretion of IL-1 β and IL-18. *Oncoimmunology* 5: e1160182.
- Lancaster, G.I., et al. 2016. PKR is not obligatory for high-fat diet-induced obesity and its associated metabolic and inflammatory complications. *Nat. Commun.* 7: 10626.
- Dalet, A., et al. 2017. Protein synthesis inhibition and GADD34 control IFN- β heterogeneous expression in response to dsRNA. *EMBO J.* 36: 761-782.
- Tanzer, M.C., et al. 2017. Combination of IAP antagonist and IFN γ activates novel caspase-10- and RIPK1-dependent cell death pathways. *Cell Death Differ.* 24: 481-491.
- Monteiro, G.E.R., et al. 2018. Mutation of adjacent cysteine residues in the NSs protein of rift valley fever virus results in loss of virulence in mice. *Virus Res.* 249: 31-44.
- Cubas-Gaona, L.L., et al. 2018. Exacerbated apoptosis of cells infected with infectious bursal disease virus (IBDV) upon exposure to Interferon α (IFN- α). *J. Virol.* 92 pii: e00364-18.

RESEARCH USE

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