

PI 3-kinase p85 α (B-9): sc-1637

BACKGROUND

Phosphatidylinositol 3-kinase (PI 3-kinase) is composed of p85 and p110 subunits. p85 lacks PI 3-kinase activity and acts as an adapter, coupling p110 to activated protein tyrosine kinase. Two forms of p85 have been described (p85 α and p85 β), each possessing one SH3 and two SH2 domains. Various p110 isoforms have been identified. p110 α and p110 β interact with p85 α , and p110 α has also been shown to interact with p85 β *in vitro*. p110 δ expression is restricted to white blood cells. It has been shown to bind p85 α and β , but it apparently does not phosphorylate these subunits. p110 δ seems to have the capacity to autophosphorylate. p110 γ does not interact with the p85 subunits. It has been shown to be activated by α and β heterotrimeric G proteins.

CHROMOSOMAL LOCATION

Genetic locus: PIK3R1 (human) mapping to 5q13.1; Pik3r1 (mouse) mapping to 13 D1.

SOURCE

PI 3-kinase p85 α (B-9) is a mouse monoclonal antibody raised against amino acids 333-430 mapping within the N-terminus SH2 domain of the 85 kDa subunit of PI 3-kinase p85 α of human origin.

PRODUCT

Each vial contains 200 μ g IgG γ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

PI 3-kinase p85 α (B-9) is available conjugated to either Alexa Fluor[®] 546 (sc-1637 AF546) or Alexa Fluor[®] 594 (sc-1637 AF594), 200 μ g/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor[®] 680 (sc-1637 AF680) or Alexa Fluor[®] 790 (sc-1637 AF790), 200 μ g/ml, for Near-Infrared (NIR) WB, IF and FCM.

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APPLICATIONS

PI 3-kinase p85 α (B-9) is recommended for detection of PI 3-kinase p85 α 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 PI 3-kinase p85 α siRNA (h): sc-36217, PI 3-kinase p85 α siRNA (m): sc-36218, PI 3-kinase p85 α siRNA (r): sc-156021, PI 3-kinase p85 α shRNA Plasmid (h): sc-36217-SH, PI 3-kinase p85 α shRNA Plasmid (m): sc-36218-SH, PI 3-kinase p85 α shRNA Plasmid (r): sc-156021-SH, PI 3-kinase p85 α shRNA (h) Lentiviral Particles: sc-36217-V, PI 3-kinase p85 α shRNA (m) Lentiviral Particles: sc-36218-V and PI 3-kinase p85 α shRNA (r) Lentiviral Particles: sc-156021-V.

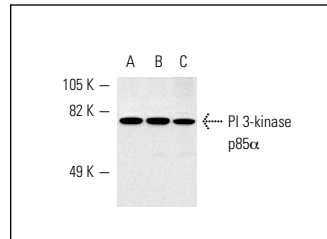
Molecular Weight of PI 3-kinase p85 α : 85 kDa.

Positive Controls: COLO 320DM cell lysate: sc-2226, Caki-1 cell lysate: sc-2224 or SW480 cell lysate: sc-2219.

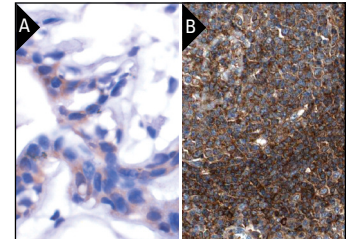
STORAGE

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

DATA



PI 3-kinase p85 α (B-9): sc-1637. Western blot analysis of PI 3-kinase p85 α expression in Caki-1 (A), COLO 320DM (B) and SW480 (C) whole cell lysates.



PI 3-kinase p85 α (B-9): sc-1637. Immunoperoxidase staining of formalin-fixed, paraffin-embedded human breast tumor showing cytoplasmic localization (A). Immunoperoxidase staining of formalin fixed, paraffin-embedded human tonsil tissue showing cytoplasmic and membrane staining of follicle and non-follicle cells and surface epithelial cells. Kindly provided by The Swedish Human Protein Atlas (HPA) program (B).

SELECT PRODUCT CITATIONS

- Widmann, C., et al. 1998. Caspase-dependent cleavage of signaling proteins during apoptosis. A turn-off mechanism for anti-apoptotic signals. *J. Biol. Chem.* 273: 7141-7147.
- Gupta, K., et al. 2018. Sorcin is involved during embryo implantation via activating VEGF/PI3K/Akt pathway in mice. *J. Mol. Endocrinol.* 60: 119-132.
- Mahajan, U.B., et al. 2018. Eplerenone pretreatment protects the myocardium against ischaemia/reperfusion injury through the phosphatidylinositol 3-kinase/Akt-dependent pathway in diabetic rats. *Mol. Cell. Biochem.* 446: 91-103.
- Sun, L., et al. 2018. NPPB modulates apoptosis, proliferation, migration and extracellular matrix synthesis of conjunctival fibroblasts by inhibiting PI3K/AKT signaling. *Int. J. Mol. Med.* 41: 1331-1338.
- Lan, D., et al. 2018. Electroacupuncture mitigates endothelial dysfunction via effects on the PI3K/Akt signalling pathway in high fat diet-induced Insulin-resistant rats. *Acupunct. Med.* 36: 162-169.
- Khaket, T.P., et al. 2018. Targeting of cathepsin C induces autophagic dysregulation that directs ER stress mediated cellular cytotoxicity in colorectal cancer cells. *Cell. Signal.* 46: 92-102.
- Chin, HK., et al. 2018. Kaempferol inhibits angiogenic ability by targeting VEGF receptor-2 and downregulating the PI3K/AKT, MEK and ERK pathways in VEGF-stimulated human umbilical vein endothelial cells. *Oncol. Rep.* 39: 2351-2357.

RESEARCH USE

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