SANTA CRUZ BIOTECHNOLOGY, INC.

p-PI 3-kinase p85α (Tyr 508): sc-12929

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

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 (p85α and p85β), each possessing one SH3 and two SH2 domains. Various p110 isoforms have been identified. p110α and p110β interact with p85α, and p110α has also been shown to interact with p85β *in vitro*. p110δ expression is restricted to white blood cells. It has been shown to bind p85α and β, but it apparently does not phosphorylate these subunits. p110δ seems to have the capacity to autophosphorylate. p110γ does not interact with the p85 subunits. It has been shown to be activated by α and βγ heterotrimeric G proteins.

CHROMOSOMAL LOCATION

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

SOURCE

p-PI 3-kinase p85α (Tyr 508) is available as either goat (sc-12929) or rabbit (sc-12929-R) polyclonal affinity purified abtibody raised against a short amino acid sequence containing Tyr-508 phosphorylated PI 3-kinase p85α of human 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-12929 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

p-PI 3-kinase p85α (Tyr 508) is recommended for detection of Tyr 508 phosphorylated PI 3-kinase p85α of mouse, rat, human and *Xenopus laevis* 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).

p-PI 3-kinase p85α (Tyr 508) is also recommended for detection of correspondingly phosphorylated PI 3-kinase p85α in additional species, including equine, canine, bovine, porcine and avian.

Suitable for use as control antibody for PI 3-kinase p85α siRNA (h): sc-36217, PI 3-kinase p85α siRNA (m): sc-36218, PI 3-kinase p85α shRNA Plasmid (h): sc-36217-SH, PI 3-kinase p85α shRNA Plasmid (m): sc-36218-SH, PI 3-kinase p85α shRNA (h) Lentiviral Particles: sc-36217-V and PI 3-kinase p85α shRNA (m) Lentiviral Particles: sc-36218-V.

Molecular Weight of p-PI 3-kinase p85α: 85 kDa.

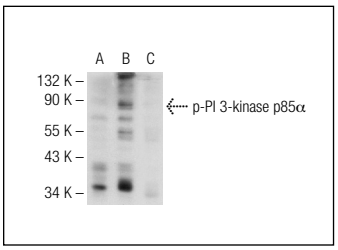
STORAGE

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

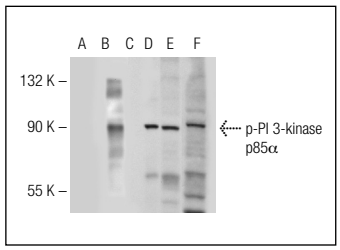
RESEARCH USE

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

DATA



p-PI 3-kinase p85α (Tyr 508)-R: sc-12929-R. Western blot analysis of PI 3-kinase p85α phosphorylation in untreated (**A**), pervanadate treated (**B**) and pervanadate and lambda protein phosphatase treated (**C**) Jurkat whole cell lysates.



Western blot analysis of PI 3-kinase p85α phosphorylation in untreated (**A**,**D**), pervanadate treated (**B**,**E**) and pervanadate and lambda protein phosphatase treated (sc-200312A) (**C**,**F**) A-431 whole cell lysates. Antibodies tested include p-PI 3-kinase p85α (Tyr 508)-R: sc-12929-R (**A**,**B**,**C**) and PI 3-kinase p85α (U13): sc-56938 (**D**,**E**,**F**).

SELECT PRODUCT CITATIONS

1. Meng, F., et al. 2003. NFκB stimulates Akt phosphorylation and gene expression by distinct signaling mechanisms. Biochim. Biophys. Acta 1630: 35-40.
2. Dutta, A., et al. 2010. Culture of K562 human myeloid leukemia cells in presence of fibronectin expresses and secretes MMP-9 in serum-free culture medium. Int. J. Clin. Exp. Pathol. 3: 288-302.
3. Sen, T., et al. 2010. Fibronectin induces matrix metalloproteinase-9 (MMP-9) in human laryngeal carcinoma cells by involving multiple signaling pathways. Biochimie 92: 1422-1434.
4. Kuo, W.T., et al. 2011. Induction of the mitochondria apoptosis pathway by phytohemagglutinin erythroagglutinating in human lung cancer cells. Ann. Surg. Oncol. 18: 848-856.
5. Heldsinger, A., et al. 2011. Synergistic interaction between leptin and cholecystokinin in the rat nodose ganglia is mediated by PI3K and STAT3 signaling pathways: implications for leptin as a regulator of short term satiety. J. Biol. Chem. 286: 11707-11715.
6. Peng, C.C., et al. 2012. Treadmill exercise improved adriamycin-induced nephropathy. J. Biol. Regul. Homeost. Agents 26: 15-28.
7. Peng, C.C., et al. 2012. Swimming exercise prevents fibrogenesis in chronic kidney disease by inhibiting the myofibroblast transdifferentiation. PLoS ONE 7: e37388.
8. Li, T.M., et al. 2012. Interleukin-11 increases cell motility and up-regulates intercellular adhesion molecule-1 expression in human chondrosarcoma cells. J. Cell. Biochem. 113: 3353-3362.
9. Chen, K.C., et al. 2014. Exercise rescued chronic kidney disease by attenuating cardiac hypertrophy through the cardiotrophin-1→LIFR/gp 130→JAK/STAT3 pathway. Eur. J. Prev. Cardiol. 21: 507-520.