SANTA CRUZ BIOTECHNOLOGY, INC.

PI 3-kinase p110δ (H-219): sc-7176

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 forms have been identified. p110α and p110β interact with p85α, and p110α has also been shown to interact with p85β *in vitro*. It has been shown to bind p85α and β, but it apparently does not phosphorylate these subunits. p110δ has the capacity to autophosphorylate and results in the nearly complete inactivation of the lipid kinase activity. Interestingly, p110γ does not interact with the p85 subunits and has been shown to be activated by α and βγ heterotrimeric G proteins. Two p110δ isoforms have been identified and are widely expressed. Isoform 1 is expressed predominantly in leukocytes while isoform 2 is expressed in normal thymus, lung and spleen tissues.

CHROMOSOMAL LOCATION

Genetic locus: PIK3CD (human) mapping to 1p36.22; Pik3cd (mouse) mapping to 4 E2.

SOURCE

PI 3-kinase p110δ (H-219) is a rabbit polyclonal antibody raised against amino acids 363-582 mapping at the N-terminus of PI 3-kinase p110δ 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.

APPLICATIONS

PI 3-kinase p110δ (H-219) is recommended for detection of PI 3-kinase p110δ of mouse, rat and 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) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for PI 3-kinase p110δ siRNA (h): sc-39131, PI 3-kinase p110δ siRNA (m): sc-39132, PI 3-kinase p110δ shRNA Plasmid (h): sc-39131-SH, PI 3-kinase p110δ shRNA Plasmid (m): sc-39132-SH, PI 3-kinase p110δ shRNA (h) Lentiviral Particles: sc-39131-V and PI 3-kinase p110δ shRNA (m) Lentiviral Particles: sc-39132-V.

Molecular Weight of PI 3-kinase p110δ isoforms: 119/33 kDa.

Positive Controls: Jurkat whole cell lysate: sc-2204, Raji whole cell lysate: sc-364236 or K-562 whole cell lysate: sc-2203.

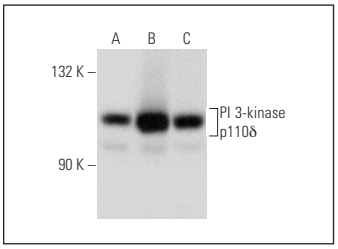
STORAGE

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



PI 3-kinase p110δ (H-219): sc-7176. Western blot analysis of PI 3-kinase p110δ expression in K-562 (**A**), Raji (**B**) and Jurkat (**C**) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Deora, A.A., et al. 1998. A redox-triggered ras-effector interaction. Recruitment of phosphatidylinositol 3'-kinase to Ras by redox stress. J. Biol. Chem. 273: 29923-29928.
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3. Fry, M.J. 2009. Phosphoinositide (PI) 3-kinase assays. Methods Mol. Biol. 462: 345-362.
4. Keck, S., et al. 2010. Activation of murine macrophages via TLR2 and TLR4 is negatively regulated by a Lyn/PI3K module and promoted by SHIP1. J. Immunol. 184: 5809-5818.
5. Acosta, Y.Y., et al. 2010. Biased binding of class IA phosphatidyl inositol 3-kinase subunits to inducible costimulator (CD278). Cell. Mol. Life Sci. 68: 3065-3079.
6. Spitzenberg, V., et al. 2010. Targeting PI3K in neuroblastoma. J. Cancer Res. Clin. Oncol. 136: 1881-1890.
7. Sun, M., et al. 2010. Cancer-derived mutations in the regulatory subunit p85α of phosphoinositide 3-kinase function through the catalytic subunit p110α. Proc. Natl. Acad. Sci. USA 107: 15547-15552.
8. Hohenester, S., et al. 2010. Phosphatidylinositol-3-kinase p110γ contributes to bile salt-induced apoptosis in primary rat hepatocytes and human hepatoma cells. J. Hepatol. 53: 918-926.
9. Geering, B., et al. 2011. A novel TNFR1-triggered apoptosis pathway mediated by class IA PI3Ks in neutrophils. Blood 117: 5953-5962.