

PI 3-kinase p110 δ (A-8): sc-55589

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 forms 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*. It has been shown to bind p85 α and β , but it apparently does not phosphorylate these subunits. p110 δ has the capacity to autophosphorylate and results in the nearly complete inactivation of the lipid kinase activity. Interestingly, p110 γ does not interact with the p85 subunits and has been shown to be activated by α and β heterotrimeric G proteins. Two p110 δ 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 p110 δ (A-8) is a mouse monoclonal antibody raised against amino acids 363-582 mapping at the N-terminus of PI 3-kinase p110 δ of human origin.

PRODUCT

Each vial contains 200 μ g IgG $_1$ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

PI 3-kinase p110 δ (A-8) is available conjugated to agarose (sc-55589 AC), 500 μ g/0.25 ml agarose in 1 ml, for IP; to HRP (sc-55589 HRP), 200 μ g/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-55589 PE), fluorescein (sc-55589 FITC), Alexa Fluor[®] 488 (sc-55589 AF488), Alexa Fluor[®] 546 (sc-55589 AF546), Alexa Fluor[®] 594 (sc-55589 AF594) or Alexa Fluor[®] 647 (sc-55589 AF647), 200 μ g/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor[®] 680 (sc-55589 AF680) or Alexa Fluor[®] 790 (sc-55589 AF790), 200 μ g/ml, for Near-Infrared (NIR) WB, IF and FCM.

APPLICATIONS

PI 3-kinase p110 δ (A-8) is recommended for detection of PI 3-kinase p110 δ of mouse, rat and human origin by Western Blotting (starting dilution 1:100, 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), 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).

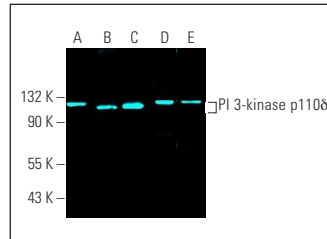
Suitable for use as control antibody for PI 3-kinase p110 δ siRNA (h): sc-39131, PI 3-kinase p110 δ siRNA (m): sc-39132, PI 3-kinase p110 δ shRNA Plasmid (h): sc-39131-SH, PI 3-kinase p110 δ shRNA Plasmid (m): sc-39132-SH, PI 3-kinase p110 δ shRNA (h) Lentiviral Particles: sc-39131-V and PI 3-kinase p110 δ shRNA (m) Lentiviral Particles: sc-39132-V.

Molecular Weight of PI 3-kinase p110 δ isoforms: 119/33 kDa.

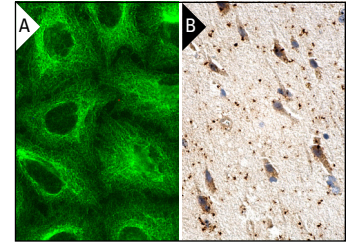
STORAGE

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 p110 δ (A-8) Alexa Fluor[®] 647: sc-55589 AF647. Direct fluorescent western blot analysis of PI 3-kinase p110 δ expression in C32 (A), Raji (B), ALL-SIL (C), ECV304 (D) and RT-4 (E) whole cell lysates. Blocked with UltraCruz[®] Blocking Reagent: sc-516214.



PI 3-kinase p110 δ (A-8): sc-55589. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoskeleton localization (A). Immunoperoxidase staining of formalin fixed, paraffin-embedded human brain tissue showing cytoplasmic staining of neuronal and glial cells (B).

SELECT PRODUCT CITATIONS

- Smith, J.A., et al. 2008. Evidence that the Nijmegen breakage syndrome protein, an early sensor of double-strand DNA breaks (DSB), is involved in HIV-1 post-integration repair by recruiting the ataxia telangiectasia-mutated kinase in a process similar to, but distinct from, cellular DSB repair. *Virology*. 5: 11.
- Owusu-Ofori, K., et al. 2013. PI3K mediates stretch-induced COX-2 expression during urinary tract obstruction. *J. Endourol.* 27: 220-229.
- Nagaraju, G.P., et al. 2014. HSP90 inhibition downregulates thymidylate synthase and sensitizes colorectal cancer cell lines to the effect of 5FU-based chemotherapy. *Oncotarget* 5: 9980-9991.
- Yuzugullu, H., et al. 2015. A PI3K p110 β -Rac signalling loop mediates Pten-loss-induced perturbation of haematopoiesis and leukaemogenesis. *Nat. Commun.* 6: 8501.
- Papaleo, F., et al. 2016. Behavioral, neurophysiological, and synaptic impairment in a transgenic neuregulin1 (NRG1-IV) murine schizophrenia model. *J. Neurosci.* 36: 4859-4875.
- Wang, B.D., et al. 2017. Alternative splicing promotes tumour aggressiveness and drug resistance in African American prostate cancer. *Nat. Commun.* 8: 15921.
- Tian, W., et al. 2018. MALAT1-miR663a negative feedback loop in colon cancer cell functions through direct miRNA-lncRNA binding. *Cell Death Dis.* 9: 857.
- Garikipati, V.N.S., et al. 2019. Circular RNA CircFndc3b modulates cardiac repair after myocardial infarction via FUS/VEGF-A axis. *Nat. Commun.* 10: 4317.

RESEARCH USE

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

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