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PTP alpha Antibody (N-term)

Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP8412a

Product Information

Application	WB, E
Primary Accession	P18433
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB0549
Calculated MW	90719
Antigen Region	89-120

Additional Information

Gene ID	5786
Other Names	Receptor-type tyrosine-protein phosphatase alpha, Protein-tyrosine phosphatase alpha, R-PTP-alpha, PTPRA, PTPA, PTPRL2
Target/Specificity	This PTP alpha antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 89-120 amino acids from the N-terminal region of human PTP alpha.
Dilution	WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	PTP alpha Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	PTPRA
Synonyms	PTPA, PTPRL2
Function	Tyrosine protein phosphatase which is involved in integrin- mediated focal adhesion formation (By similarity). Following integrin engagement, specifically

	recruits BCAR3, BCAR1 and CRK to focal adhesions thereby promoting SRC-mediated phosphorylation of BRAC1 and the subsequent activation of PAK and small GTPase RAC1 and CDC42 (By similarity).
Cellular Location	Cell membrane; Single-pass type I membrane protein. Cell junction, focal adhesion {ECO:0000250|UniProtKB:P18052}. Note=Localizes to focal adhesion sites following integrin engagement. {ECO:0000250|UniProtKB:P18052}

Background

Phosphorylation of receptors by protein kinases is a process that can be reversed by a group of enzymes called protein phosphatases. Coordinated control of kinases and phosphatases provides the cell with the capacity to rapidly switch between phosphorylated and dephosphorylated protein states in dynamic response to environmental stimuli. Activation of critical enzymes by kinase phosphorylation alone is not enough to provide adequate regulation ?it is the combination with phosphatase dephosphorylation that effectively creates on/off switches to control cellular events. Errors in control, either through kinases or their counterpart phosphatases, can lead to unchecked cell growth attributable to human cancers and developmental disorders. Potential mechanisms to control dephosphorylation include changes in the expression of protein phosphatases, their subcellular localization, phosphorylation of phosphatase catalytic and regulatory subunits and regulation by endogenous phosphatase inhibitors. Most protein phosphatases are not stringently specific for their substrates. Consequently, changes in phosphatase activity may have a broad impact on dephosphorylation and turnover of phosphoproteins that are substrates for different kinases. This may be an important point of control to connect cellular circuitry of interrelated signaling pathways, and to synchronize physiological responses.

References

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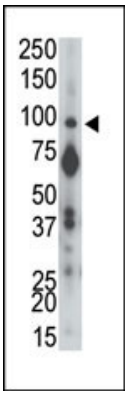
Kaplan, R., et al., Proc. Natl. Acad. Sci. U.S.A. 87(18):7000-7004 (1990).
Krueger, N.X., et al., EMBO J. 9(10):3241-3252 (1990).
Sap, J., et al., Proc. Natl. Acad. Sci. U.S.A. 87(16):6112-6116 (1990).
Jirik, F.R., et al., FEBS Lett. 273 (1-2), 239-242 (1990).

Images



The anti-PTPalpha N-term Pab (Cat. #AP8412a) is used in Western blot to detect PTPalpha in mouse brain tissue lysate.