

**PTP zeta Antibody (Center) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP8422a**

**Specification**

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**PTP zeta Antibody (Center) Blocking peptide - Product Information**

Primary Accession [P23471](#)

**PTP zeta Antibody (Center) Blocking peptide - Additional Information**

**Gene ID** 5803

**Other Names**

Receptor-type tyrosine-protein phosphatase zeta, R-PTP-zeta, Protein-tyrosine phosphatase receptor type Z polypeptide 1, Protein-tyrosine phosphatase receptor type Z polypeptide 2, R-PTP-zeta-2, PTPRZ1, HTPZP2, PTPRZ, PTPRZ2, PTPZ

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP8422a](/product/products/AP8422a) was selected from the Center region of human PTPzeta . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**PTP zeta Antibody (Center) Blocking peptide - Protein Information**

**Name** PTPRZ1

**Synonyms** HTPZP2, PTPRZ, PTPRZ2, PTPZ

**Function**

Protein tyrosine phosphatase that negatively regulates oligodendrocyte precursor proliferation in the embryonic spinal cord. Required for normal differentiation of the precursor cells into mature, fully myelinating oligodendrocytes. May play a role in protecting oligodendrocytes against apoptosis. May play a role in the establishment of contextual memory, probably via the dephosphorylation of proteins that are part of important signaling cascades (By similarity).

**Cellular Location**

[Isoform 1]: Cell membrane; Single-pass type I membrane protein. Secreted. Note=A secreted

form is apparently generated by shedding of the extracellular domain

#### **Tissue Location**

Specifically expressed in the central nervous system, where it is localized in the Purkinje cell layer of the cerebellum, the dentate gyrus, and the subependymal layer of the anterior horn of the lateral ventricle. Developmentally regulated in the brain.

#### **PTP zeta Antibody (Center) Blocking peptide - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

#### **PTP zeta Antibody (Center) Blocking peptide - Images**

#### **PTP zeta Antibody (Center) Blocking peptide - 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.

#### **PTP zeta Antibody (Center) Blocking peptide - References**

Muller, S., et al., Oncogene 22(43):6661-6668 (2003). Harroch, S., et al., Nat. Genet. 32(3):411-414 (2002). Kawachi, H., et al., Proc. Natl. Acad. Sci. U.S.A. 98(12):6593-6598 (2001). Thomaidou, D., et al., J. Neurochem. 78(4):767-778 (2001). Meng, K., et al., Proc. Natl. Acad. Sci. U.S.A. 97(6):2603-2608 (2000).