

**LyPTP Antibody (C-term) Blocking Peptide**  
**Synthetic peptide**  
**Catalog # BP8406b****Specification**

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**LyPTP Antibody (C-term) Blocking Peptide - Product Information**Primary Accession [Q9Y2R2](#)**LyPTP Antibody (C-term) Blocking Peptide - Additional Information**

Gene ID 26191

**Other Names**

Tyrosine-protein phosphatase non-receptor type 22, Hematopoietic cell protein-tyrosine phosphatase 70Z-PEP, Lymphoid phosphatase, LyP, PEST-domain phosphatase, PEP, PTPN22, PTPN8

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP8406b](/product/products/AP8406b) was selected from the C-term region of human LyPTP. 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.

**LyPTP Antibody (C-term) Blocking Peptide - Protein Information**

Name PTPN22

Synonyms PTPN8

**Function**

Acts as a negative regulator of T-cell receptor (TCR) signaling by direct dephosphorylation of the Src family kinases LCK and FYN, ITAMs of the TCRz/CD3 complex, as well as ZAP70, VAV, VCP and other key signaling molecules (PubMed: [16461343](http://www.uniprot.org/citations/16461343), PubMed: [18056643](http://www.uniprot.org/citations/18056643)). Associates with and probably dephosphorylates CBL. Dephosphorylates LCK at its activating 'Tyr-394' residue (PubMed: [21719704](http://www.uniprot.org/citations/21719704)). Dephosphorylates ZAP70 at its activating 'Tyr-493' residue (PubMed: [21719704](#)).

[16461343](http://www.uniprot.org/citations/16461343)). Dephosphorylates the immune system activator SKAP2 (PubMed:[21719704](http://www.uniprot.org/citations/21719704)). Positively regulates toll-like receptor (TLR)-induced type 1 interferon production (PubMed:[23871208](http://www.uniprot.org/citations/23871208)). Promotes host antiviral responses mediated by type 1 interferon (By similarity). Regulates NOD2-induced pro-inflammatory cytokine secretion and autophagy (PubMed:[23991106](http://www.uniprot.org/citations/23991106)). Acts as an activator of NLRP3 inflammasome assembly by mediating dephosphorylation of 'Tyr-861' of NLRP3 (PubMed:[27043286](http://www.uniprot.org/citations/27043286)). Dephosphorylates phospho-anandamide (p-AEA), an endocannabinoid to anandamide (also called N-arachidonoyl ethanolamide) (By similarity).

#### **Cellular Location**

Cytoplasm {ECO:0000250|UniProtKB:P29352}.

#### **Tissue Location**

Expressed in bone marrow, B and T-cells, PBMCs, natural killer cells, monocytes, dendritic cells and neutrophils (PubMed:15208781). Both isoform 1 and 4 are predominantly expressed in lymphoid tissues and cells. Isoform 1 is expressed in thymocytes and both mature B and T-cells.

### **LyPTP Antibody (C-term) Blocking Peptide - Protocols**

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

- [Blocking Peptides](#)

### **LyPTP Antibody (C-term) Blocking Peptide - Images**

### **LyPTP Antibody (C-term) 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.

### **LyPTP Antibody (C-term) Blocking Peptide - References**

Cohen, S., et al., Blood 93(6):2013-2024 (1999).