

## CD79A (Phospho Tyr182) Rabbit pAb

CatalogNo: YP1292

### Key Features

#### Host Species

- Rabbit

#### Reactivity

- Human, Mouse, Rat

#### Applications

- WB

#### MW

- 25kD (Observed)

#### Isotype

- IgG

### Storage

**Storage\*** -15°C to -25°C/1 year (Do not lower than -25°C)

**Formulation** Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.

### Recommended Dilution Ratios

WB 1:1000-2000

### Basic Information

**Clonality** Polyclonal

### Immunogen Information

**Immunogen** Synthesized phospho peptide around human CD79A (Tyr182)

**Specificity** This antibody detects endogenous levels of Human CD79A (phospho-Tyr182)

### Target Information

**Gene name** CD79A IGA MB1

**Protein Name**

CD79A (Tyr182)

Organism	Gene ID	UniProt ID
Human	<a href="#">973</a> ;	<a href="#">P11912</a> ;
Mouse	<a href="#">12518</a> ;	<a href="#">P11911</a> ;

**Cellular Localization**

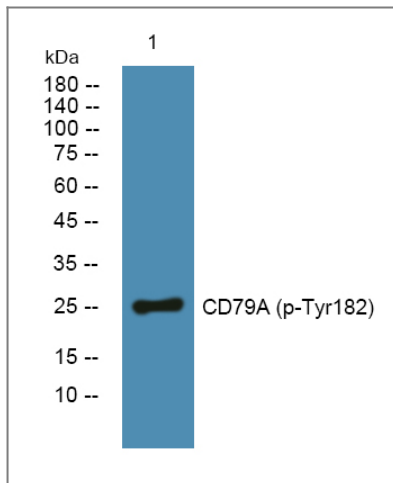
Cell membrane; Single-pass type I membrane protein. Following antigen binding , the BCR has been shown to translocate from detergent-soluble regions of the cell membrane to lipid rafts although signal transduction through the complex can also occur outside lipid rafts. .

**Tissue specificity** B-cells.

**Function**

Disease:Defects in CD79A are a cause of non-Bruton type agammaglobulinemia [MIM:601495]. Agammaglobulinemia is an immunodeficiency disease which results in developmental defects in the maturation pathway of B-cells. Two different mutations , one at the splice donor site of intron 2 and the other at the splice acceptor site for exon 3 , have been identified. Both mutations give rise to a truncated protein. ,Function:Required in cooperation with CD79B for initiation of the signal transduction cascade activated by binding of antigen to the B-cell antigen receptor complex (BCR) which leads to internalization of the complex , trafficking to late endosomes and antigen presentation. Also required for BCR surface expression and for efficient differentiation of pro- and pre-B-cells. Stimulates SYK autophosphorylation and activation. Binds to BLNK , bringing BLNK into proximity with SYK and allowing SYK to phosphorylate BLNK. Also interacts with and increases activity of some Src-family tyrosine kinases. Represses BCR signaling during development of immature B cells. ,online information:CD79A mutation db ,PTM:Phosphorylated on tyrosine , serine and threonine residues upon B-cell activation. Phosphorylation of tyrosine residues by Src-family kinases is an early and essential feature of the BCR signaling cascade. The phosphorylated tyrosines serve as docking sites for SH2-domain containing kinases , leading to their activation which in turn leads to phosphorylation of downstream targets. Phosphorylation of serine and threonine residues may prevent subsequent tyrosine phosphorylation. ,similarity:Contains 1 Ig-like C2-type (immunoglobulin-like) domain. ,similarity:Contains 1 ITAM domain. ,subcellular location:Following antigen binding , the BCR has been shown to translocate from detergent-soluble regions of the cell membrane to lipid rafts although signal transduction through the complex can also occur outside lipid rafts. ,subunit:Heterodimer of alpha and beta chains; disulfide-linked. Part of the B-cell antigen receptor complex where the alpha/beta chain heterodimer is non-covalently associated with an antigen-specific membrane-bound surface immunoglobulin of two heavy chains and two light chains. Interacts through its phosphorylated ITAM domain with the SH2 domains of SYK which stimulates SYK autophosphorylation and activation. Also interacts , when phosphorylated on Tyr-210 , with the SH2 domain of BLNK/SLP65 , bringing BLNK into proximity with SYK and allowing SYK to phosphorylate BLNK which is necessary for trafficking of the BCR to late endosomes. Interacts with Src-family tyrosine kinases including FYN and LYN , increasing their activity. ,tissue specificity:B-cells. ,

**Validation Data**



Western blot analysis of lysates from KB cells, primary antibody was diluted at 1:1000, 4°C over night

## Contact information

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