

## BTK (Phospho Ser179) Rabbit pAb

CatalogNo: YP1838

### | Key Features

#### Host Species

- Rabbit

#### Reactivity

- Human, Mouse

#### Applications

- IHC, WB

#### MW

- 80kD (Observed)

### | Recommended Dilution Ratios

WB 1:500-2000

IHC 1:50-200

### | Storage

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

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

### | Basic Information

**Clonality** Polyclonal

### | Immunogen Information

**Immunogen** Synthesized peptide derived from human BTK (Phospho Ser179)

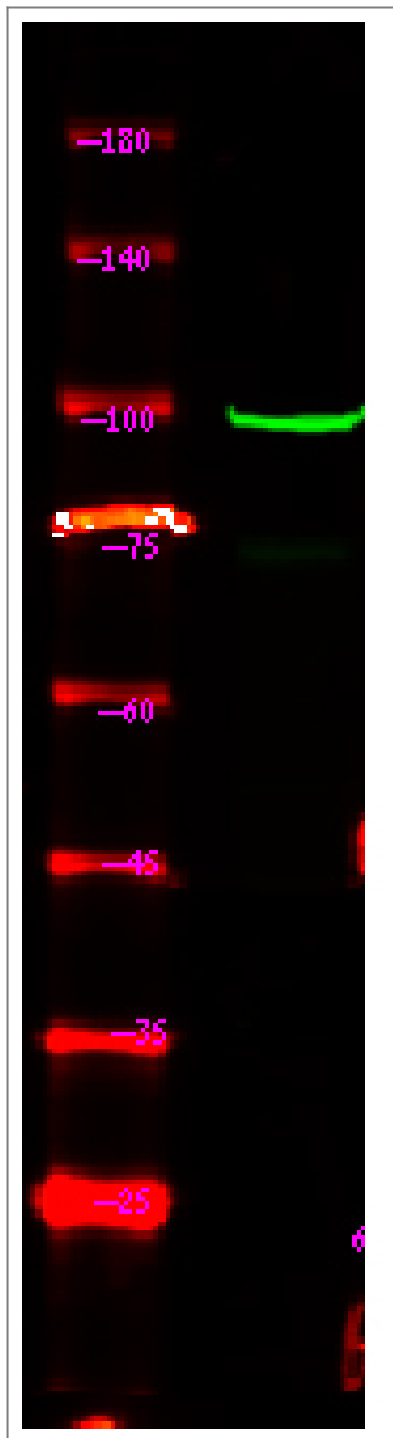
**Specificity** This antibody detects endogenous levels of BTK (Phospho Ser179) Rabbit pAb at Human, Mouse

### | Target Information

**Gene name** BTK AGMX1 ATK BPK

<b>Protein Name</b>	Tyrosine-protein kinase BTK (Agammaglobulinaemia tyrosine kinase) (ATK) (B-cell progenitor kinase) (BPK) (Bruton tyrosine kinase)		
	<b>Organism</b>	<b>Gene ID</b>	<b>UniProt ID</b>
	Human	<a href="#">695;</a>	<a href="#">Q06187;</a>
	Mouse	<a href="#">12229;</a>	<a href="#">P35991;</a>
<b>Cellular Localization</b>	Cytoplasm. Cell membrane; Peripheral membrane protein. Nucleus. In steady state, BTK is predominantly cytosolic. Following B-cell receptor (BCR) engagement by antigen, translocates to the plasma membrane through its PH domain. Plasma membrane localization is a critical step in the activation of BTK. A fraction of BTK also shuttles between the nucleus and the cytoplasm, and nuclear export is mediated by the nuclear export receptor CRM1.		
<b>Tissue specificity</b>	Predominantly expressed in B-lymphocytes.		
<b>Function</b>	<p>Catalytic activity:ATP + a [protein]-L-tyrosine = ADP + a [protein]-L-tyrosine phosphate.,cofactor:Binds 1 zinc ion per subunit.,Disease:Defects in BTK are the cause of X-linked agammaglobulinemia (XLA) [MIM:300755]; also called X-linked agammaglobulinemia type 1 (AGMX1) or immunodeficiency type 1 (IMD1). XLA is a humoral immunodeficiency disease which results in developmental defects in the maturation pathway of B-cells. Affected boys have normal levels of pre-B-cells in their bone marrow but virtually no circulating mature B-lymphocytes. This results in a lack of immunoglobulins of all classes and leads to recurrent bacterial infections like otitis, conjunctivitis, dermatitis, sinusitis in the first few years of life, or even some patients present overwhelming sepsis or meningitis, resulting in death in a few hours. Treatment in most cases is by infusion of intravenous immunoglobulin.,Disease:Defects in BTK may be the cause of X-linked hypogammaglobulinemia and isolated growth hormone deficiency (XLA-IGHD) [MIM:307200]; also known as agammaglobulinemia and isolated growth hormone deficiency or Fleisher syndrome or isolated growth hormone deficiency type 3 (IGHD3). In rare cases XLA is inherited together with isolated growth hormone deficiency (IGHD).,enzyme regulation:Inhibited by IBTK. Activated by phosphorylation.,Function:Plays a crucial role in B-cell ontogeny. Transiently phosphorylates GTF2I on tyrosine residues in response to B-cell receptor cross-linking. Required for the formation of functional ARID3A DNA-binding complexes.,online information:BTK mutation db,PTM:Autophosphorylated on Tyr-223 and Tyr-551. Phosphorylation of Tyr-223 may create a docking site for a SH2 containing protein.,similarity:Belongs to the protein kinase superfamily. Tyr protein kinase family.,similarity:Belongs to the protein kinase superfamily. Tyr protein kinase family. TEC subfamily.,similarity:Contains 1 Btk-type zinc finger.,similarity:Contains 1 PH domain.,similarity:Contains 1 protein kinase domain.,similarity:Contains 1 SH2 domain.,similarity:Contains 1 SH3 domain.,subunit:Binds GTF2I through the PH domain. Interacts with SH3BP5 via the SH3 domain. Interacts with IBTK via its PH domain. Interacts with GTF2I and ARID3A.,</p>		

| Validation Data



Western Blot analysis of mouse heart ,using primary antibody at 1:1000 dilution. Secondary antibody(catalog#:RS23920) was diluted at 1:10000

## Contact information

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Ser179) Rabbit pAb**

