Best Tools for immunology Research

Btk (Phospho Ser180) rabbit pAb

Catalog No :	YP1279
Reactivity :	Human;Rat;Mouse;
Applications :	WB
Target :	Btk
Fields :	>>NF-kappa B signaling pathway;>>Osteoclast differentiation;>>Platelet activation;>>B cell receptor signaling pathway;>>Fc epsilon RI signaling pathway;>>Epstein-Barr virus infection;>>Primary immunodeficiency
Gene Name :	BTK AGMX1 ATK BPK
Protein Name :	Btk (Ser180)
Human Gene Id :	695
Human Swiss Prot No :	Q06187
Mouse Gene Id :	12229
Mouse Swiss Prot No :	P35991
Immunogen :	Synthesized phosho peptide around human Btk (Ser180)
Specificity :	This antibody detects endogenous levels of Human Btk (phospho-Ser180)
Formulation :	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Source :	Polyclonal, Rabbit,IgG
Dilution :	WB 1:1000-2000
Purification :	The antibody was affinity-purified from rabbit serum by affinity-chromatography using specific immunogen.
Concentration :	1 mg/ml

Storage Stability :	-15°C to -25°C/1 year(Do not lower than -25°C)
Observed Band :	80kD
Cell Pathway :	B_Cell_Antigen;Fc epsilon RI;Primary immunodeficiency;
Background :	The protein encoded by this gene plays a crucial role in B-cell development. Mutations in this gene cause X-linked agammaglobulinemia type 1, which is an immunodeficiency characterized by the failure to produce mature B lymphocytes, and associated with a failure of Ig heavy chain rearrangement. Alternative splicing results in multiple transcript variants encoding different isoforms. [provided by RefSeq, Dec 2013],
Function :	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.,
Subcellular Location :	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.
Expression :	Predominantly expressed in B-lymphocytes.

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