

## **Btk Polyclonal Antibody**

Catalog No: YT5346

**Reactivity:** Human; Mouse; Rat

**Applications:** WB;IHC;IF;ELISA

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

**Protein Name:** Tyrosine-protein kinase BTK

Q06187

P35991

Human Gene Id: 695

**Human Swiss Prot** 

No:

Mouse Gene Id: 12229

**Mouse Swiss Prot** 

No:

**Immunogen:** Synthesized peptide derived from Btk . at AA range: 490-570

**Specificity:** Btk Polyclonal Antibody detects endogenous levels of Btk protein only when non-

phosphorylation at Y550.

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

Source: Polyclonal, Rabbit, IgG

**Dilution :** WB 1:500 - 1:2000. IHC 1:100 - 1:300. ELISA: 1:20000.. IF 1:50-200

**Purification:** The antibody was affinity-purified from rabbit antiserum by affinity-

chromatography using epitope-specific immunogen.



**Concentration:** 1 mg/ml

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

Observed Band: 76kD

B\_Cell\_Antigen;Fc epsilon RI;Primary immunodeficiency; **Cell Pathway:** 

The protein encoded by this gene plays a crucial role in B-cell development. **Background:** 

> 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.,

Cytoplasm. Cell membrane; Peripheral membrane protein. Nucleus. In steady Subcellular Location:

state, BTK is predominantly cytosolic. Following B-cell receptor (BCR)

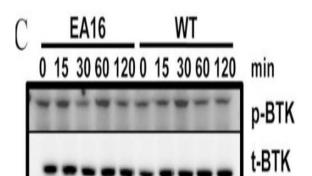
engagement by antigen, translocates to the plasma membrane through its PH domain. Plasma membrane localization is a critical step in the activation of BTK. A fraction of BTK also shuttles between the nucleus and the cytoplasm, and

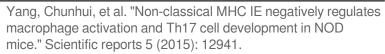
nuclear export is mediated by the nuclear export receptor CRM1.

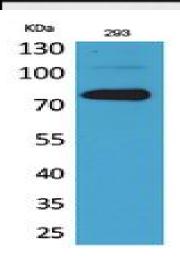
Predominantly expressed in B-lymphocytes. **Expression:** 

## **Products Images**









Western Blot analysis of 293 cells using Btk Polyclonal Antibody. Secondary antibody(catalog#:RS0002) was diluted at 1:20000