

**I $\kappa$ B- $\beta$  Polyclonal Antibody**  
Catalog # AP70604**Specification**

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**I $\kappa$ B- $\beta$  Polyclonal Antibody - Product Information**

Application	<b>WB</b>
Primary Accession	<a href="#">Q15653</a>
Reactivity	<b>Human, Mouse</b>
Host	<b>Rabbit</b>
Clonality	<b>Polyclonal</b>

**I $\kappa$ B- $\beta$  Polyclonal Antibody - Additional Information****Gene ID** 4793**Other Names**NFKBIB; IKBB; TRIP9; NF-kappa-B inhibitor beta; NF-kappa-BIB; I-kappa-B-beta; I $\kappa$ B-B; I $\kappa$ B-beta; I $\kappa$ appabeta; Thyroid receptor-interacting protein 9; TR-interacting protein 9; TRIP-9**Dilution**

WB~~Western Blot: 1/500 - 1/2000. ELISA: 1/5000. Not yet tested in other applications.

**Format**

Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.09% (W/V) sodium azide.

**Storage Conditions**

-20°C

**I $\kappa$ B- $\beta$  Polyclonal Antibody - Protein Information****Name** NFKBIB**Synonyms** IKBB, TRIP9**Function**

Inhibits NF-kappa-B by complexing with and trapping it in the cytoplasm. However, the unphosphorylated form resynthesized after cell stimulation is able to bind NF-kappa-B allowing its transport to the nucleus and protecting it to further NFKBIA-dependent inactivation. Association with inhibitor kappa B-interacting NKIRAS1 and NKIRAS2 prevent its phosphorylation rendering it more resistant to degradation, explaining its slower degradation.

**Cellular Location**

Cytoplasm. Nucleus.

**Tissue Location**

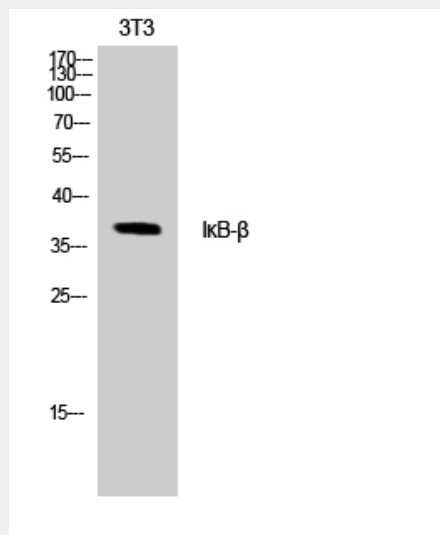
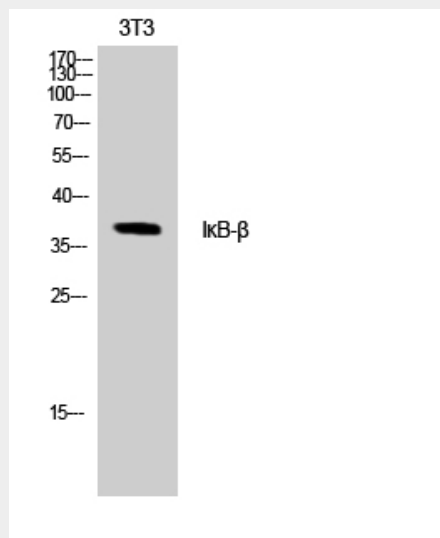
Expressed in all tissues examined.

## **I $\kappa$ B- $\beta$ Polyclonal Antibody - Protocols**

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

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

## **I $\kappa$ B- $\beta$ Polyclonal Antibody - Images**



## **I $\kappa$ B- $\beta$ Polyclonal Antibody - Background**

Inhibits NF-kappa-B by complexing with and trapping it in the cytoplasm. However, the

unphosphorylated form resynthesized after cell stimulation is able to bind NF-kappa-B allowing its transport to the nucleus and protecting it to further NFKBIA- dependent inactivation. Association with inhibitor kappa B- interacting NKIRAS1 and NKIRAS2 prevent its phosphorylation rendering it more resistant to degradation, explaining its slower degradation.