

# IκB-β (phospho Thr19) Polyclonal Antibody

Catalog # AP67232

#### Specification

## IκB-β (phospho Thr19) Polyclonal Antibody - Product Information

Application Primary Accession Reactivity Host Clonality WB, IHC-P 015653 Human Rabbit Polyclonal

#### IκB-β (phospho Thr19) Polyclonal Antibody - Additional Information

Gene ID 4793

**Other Names** NFKBIB; IKBB; TRIP9; NF-kappa-B inhibitor beta; NF-kappa-BIB; I-kappa-B-beta; IkB-B; IkB-beta; IkappaBbeta; Thyroid receptor-interacting protein 9; TR-interacting protein 9; TRIP-9

Dilution WB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. ELISA: 1/5000. Not yet tested in other applications. IHC-P~~N/A

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

**Storage Conditions** -20°C

#### IκB-β (phospho Thr19) 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.

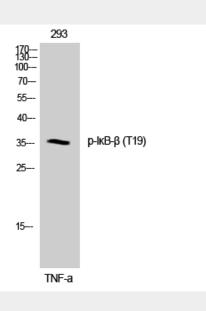


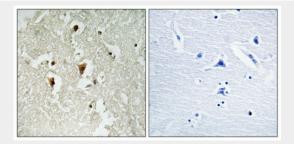
## IκB-β (phospho Thr19) Polyclonal Antibody - Protocols

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

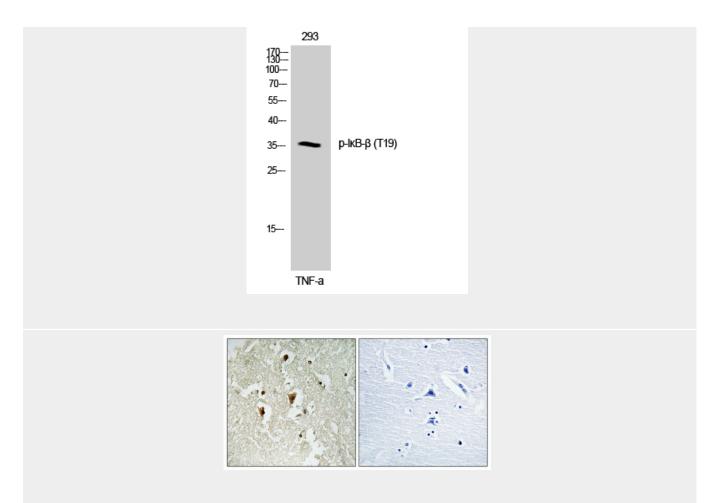
- <u>Western Blot</u>
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- <u>Flow Cytomety</u>
- <u>Cell Culture</u>

#### IκB-β (phospho Thr19) Polyclonal Antibody - Images









# IκB-β (phospho Thr19) 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.