

IκB-β (phospho Ser23) Polyclonal Antibody
Catalog # AP67085**Specification**

IκB-β (phospho Ser23) Polyclonal Antibody - Product Information

Application	WB, IHC-P, IF
Primary Accession	Q15653
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal

IκB-β (phospho Ser23) Polyclonal Antibody - Additional Information**Gene ID** 4793**Other Names**

NFKBIB; IKBB; TRIP9; NF-kappa-B inhibitor beta; NF-kappa-BIB; I-kappa-B-beta; IκB-B; IκB-beta; IκappaBbeta; Thyroid receptor-interacting protein 9; TR-interacting protein 9; TRIP-9

DilutionWB~~Western Blot: 1/500 - 1/2000. Immunohistochemistry: 1/100 - 1/300. Immunofluorescence: 1/200 - 1/1000. ELISA: 1/40000. Not yet tested in other applications.
IHC-P~~N/A
IF~~1:50~200**Format**

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

Storage Conditions

-20°C

IκB-β (phospho Ser23) 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

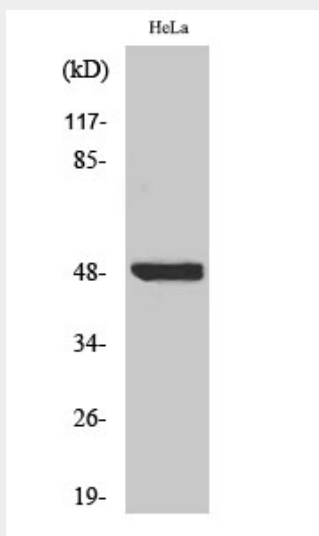
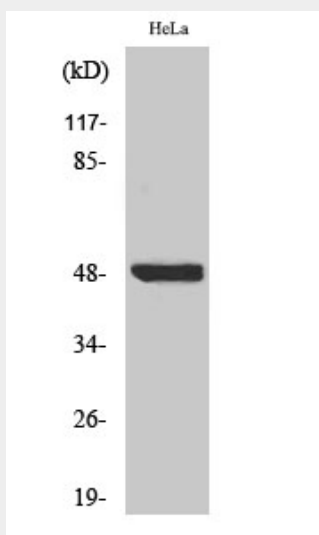
Expressed in all tissues examined.

I κ B- β (phospho Ser23) 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 κ B- β (phospho Ser23) Polyclonal Antibody - Images



I κ B- β (phospho Ser23) 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.