

**Anti-I $\kappa$ B $\alpha$  (C-terminus) Antibody**  
**Catalog # AN1814****Specification****Anti-I $\kappa$ B $\alpha$  (C-terminus) Antibody - Product Information**

Primary Accession	<a href="#">P25963</a>
Reactivity	<b>Bovine</b>
Host	<b>Rabbit</b>
Clonality	<b>Rabbit Polyclonal</b>
Isotype	<b>IgG</b>
Calculated MW	<b>35609</b>

**Anti-I $\kappa$ B $\alpha$  (C-terminus) Antibody - Additional Information**Gene ID **4792****Other Names**I $\kappa$ B, MAD3, I $\kappa$ appaB $\alpha$ , NF $\kappa$ B inhibitor I $\kappa$ B $\alpha$ **Target/Specificity**

The NF- $\kappa$ B/Rel transcription factors are present in the cytosol in an inactive state complexed with the inhibitory I $\kappa$ B proteins. Activation of I $\kappa$ B $\alpha$  occurs through both serine and tyrosine phosphorylation events. Activation through phosphorylation at Ser-32 and Ser-36 is followed by proteasome-mediated degradation, resulting in the release and nuclear translocation of active NF- $\kappa$ B. This pathway of I $\kappa$ B $\alpha$  regulation occurs in response to various NF- $\kappa$ B-activating agents, such as TNF $\alpha$ , interleukins, LPS, and irradiation. An alternative pathway for I $\kappa$ B $\alpha$  regulation occurs through tyrosine phosphorylation of Tyr-42 and Tyr-305. Tyr-42 is phosphorylated in response to oxidative stress and growth factors. This phosphorylation can lead to degradation of I $\kappa$ B $\alpha$  and NF- $\kappa$ B-activation. In contrast, Tyr-305 phosphorylation by c-Abl has been implicated in I $\kappa$ B $\alpha$  nuclear translocation and inhibition of NF- $\kappa$ B-activation. Thus, tyrosine phosphorylation of I $\kappa$ B $\alpha$  may be an important regulatory mechanism in NF- $\kappa$ B signaling.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**

Anti-I $\kappa$ B $\alpha$  (C-terminus) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**Shipping**

Blue Ice

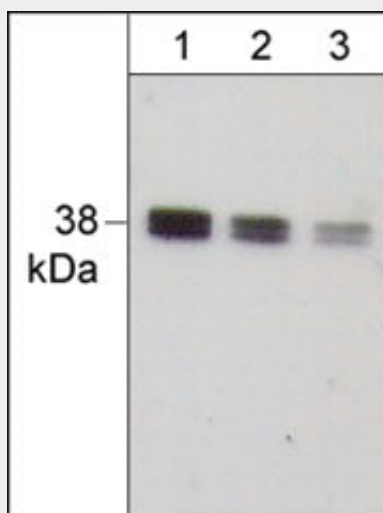
**Anti-I $\kappa$ B $\alpha$  (C-terminus) Antibody - Protocols**

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](#)

#### Anti-I $\kappa$ B $\alpha$ (C-terminus) Antibody - Images



Western blot image of human A431. The Blots were probed with anti-I $\kappa$ B $\alpha$  (C-term.) polyclonal antibody at a dilution of 1:500 (lane 1), 1:1000 (lane 2), and 1:2000 (lane 3).

#### Anti-I $\kappa$ B $\alpha$ (C-terminus) Antibody - Background

The NF- $\kappa$ B/Rel transcription factors are present in the cytosol in an inactive state complexed with the inhibitory I $\kappa$ B proteins. Activation of I $\kappa$ B $\alpha$  occurs through both serine and tyrosine phosphorylation events. Activation through phosphorylation at Ser-32 and Ser-36 is followed by proteasome-mediated degradation, resulting in the release and nuclear translocation of active NF- $\kappa$ B. This pathway of I $\kappa$ B $\alpha$  regulation occurs in response to various NF- $\kappa$ B-activating agents, such as TNF $\alpha$ , interleukins, LPS, and irradiation. An alternative pathway for I $\kappa$ B $\alpha$  regulation occurs through tyrosine phosphorylation of Tyr-42 and Tyr-305. Tyr-42 is phosphorylated in response to oxidative stress and growth factors. This phosphorylation can lead to degradation of I $\kappa$ B $\alpha$  and NF- $\kappa$ B-activation. In contrast, Tyr-305 phosphorylation by c-Abl has been implicated in I $\kappa$ B $\alpha$  nuclear translocation and inhibition of NF- $\kappa$ B-activation. Thus, tyrosine phosphorylation of I $\kappa$ B $\alpha$  may be an important regulatory mechanism in NF- $\kappa$ B signaling.

#### Anti-I $\kappa$ B $\alpha$ (C-terminus) Antibody - Citations

- [Factor L2 ameliorates the Progression of K/BxN Serum-Induced Arthritis by Blocking TLR7 Mediated IRAK4/IKK \$\beta\$ /IRF5 and NF- \$\kappa\$ B Signaling Pathways](#)