

Anti-IκBα (Tyr-305), Phosphospecific Antibody

Catalog # AN1817

Product Information

Application WB
Primary Accession P25963
Host Rabbit

Clonality Rabbit Polyclonal

Isotype IgG **Calculated MW** 35609

Additional Information

Gene ID 4792

Other Names IkB, MAD3, IkappaBalpha, NFkappaB inhibitor IkBa

Target/Specificity The NF-κB/Rel transcription factors are present in the cytosol in an inactive

state complexed with the inhibitory IkB proteins. Activation of IkB α 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-kB. This pathway of IkB α regulation occurs in response to various NF-kB-activating agents, such as TNF α , interleukins, LPS, and irradiation. An

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 IκBα and NF-κB-activation. In contrast, Tyr-305

alternative pathway for IkBa regulation occurs through tyrosine

phosphorylation by c-Abl has been implicated in IkB α nuclear translocation and inhibition of NF-kB-activation. Thus, tyrosine phosphorylation of IkB α

may be an important regulatory mechanism in NF-κB signaling.

Dilution WB~~1:1000

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-IκBα (Tyr-305), Phosphospecific Antibody is for research use only and not

for use in diagnostic or therapeutic procedures.

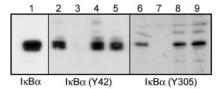
Shipping Blue Ice

Background

The NF- κ B/Rel transcription factors are present in the cytosol in an inactive state complexed with the inhibitory IkB proteins. Activation of IkB α 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- κ B. This pathway of IkB α regulation occurs in

response to various NF-κB-activating agents, such as TNFα, interleukins, LPS, and irradiation. An alternative pathway for IκBα 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 IκBα and NF-κB-activation. In contrast, Tyr-305 phosphorylation by c-Abl has been implicated in IκBα nuclear translocation and inhibition of NF-κB-activation. Thus, tyrosine phosphorylation of IκBα may be an important regulatory mechanism in NF-κB signaling.

Images



Western blot analysis of A431 cells treated with pervanadate (1 mM) for 30 min. Blots were probed with anti-IkB α (lane 1), anti-IkB α (Tyr-42) (IP1031; lanes 2-5), or anti-IkB α (Tyr-305) (AN1817; lanes 6-9). In some lanes, the antibodies were used in the absence (lane 2 & 6) or presence of IkB α (Tyr-42) (lane 3 & 8) or IkB α (Tyr-305) (lane 4 & 7) blocking peptides, or BSA conjugated to phospho-tyrosine (lane 5 & 9).

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.