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Anti-IκBα (Ser-32/Ser-36), Phosphospecific Antibody

Catalog # AN1815

Product Information

Application	WB, IP
Primary Accession	P25963
Host	Mouse
Clonality	Mouse Monoclonal
Isotype	IgG1
Clone Names	39A1413
Calculated MW	35609

Additional Information

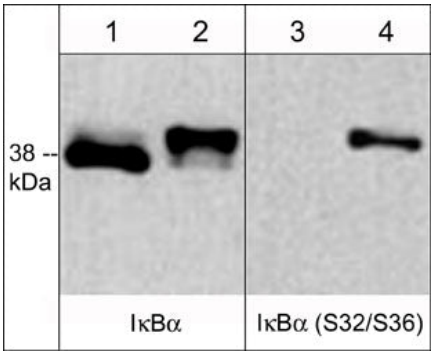
Gene ID	4792
Other Names	IkB, MAD3, IkappaBalpha, NFkappaB inhibitor IkBa
Target/Specificity	The NF-κB/Rel transcription factors are present in the cytosol in an inactive state complexed with the inhibitory IκB proteins. Activation of IκBα occurs through both serine and tyrosine phosphorylation events. 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-κB. This pathway of IκBα 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.
Dilution	WB~~1:1000 IP~~N/A
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 (Ser-32/Ser-36), Phosphospecific Antibody is for research use only and not for use in diagnostic or therapeutic procedures.
Shipping	Blue Ice

Background

The NF-κB/Rel transcription factors are present in the cytosol in an inactive state complexed with the inhibitory IκB proteins. Activation of IκBα 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-κB. This pathway of IκBα 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 Jurkat cells untreated (lanes 1 & 3) or treated with TNFα (1 nM). The blots were probed with anti-IκBα (lanes 1 & 2) or anti-IκBα (Ser-32/Ser-36) (lanes 3 & 4).