Data Sheet (Cat.No.TMAY-02203H)

Anti-IkB alpha/NFKBIA Antibody-HRP (6U300)

Product Details

Ig Type:	Mouse IgG2b
Reactivity:	Human
Conjugation:	HRP
Clone:	6U300
Purification:	Protein A

Applications

Application:	ELISA
Recommended	ELISA: 0.1-1 μg/ml

Properties

Stability & Storage:	Store at 2°C-8°C for 1 month. Store at -20°C or -80°C for 12 months. Avoid repeated freeze-thaw cycles. Preservative-Free. Keep away from direct sunlight.
Shipping:	Shipping with blue ice.

Antigen Details

Immunogen:	Recombinant Protein: Human IkB alpha / NFKBIA protein (TMPY-01710)
Antigen Species:	Human
Synonyms:	MAD-3;IKBA;nuclear factor of κ light polypeptide gene enhancer in B-cells inhibitor, alpha;IkB α;nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor, alpha;nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor, α;NFKBI

Research Background

Nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor, alpha (IkB alpha, NFKBIA, or IKBA), is a member of the NF-kappa-B inhibitor family that function to inhibit the NF-kB transcription factor. NFKBIA inhibits NF-kB by masking the nuclear localization signals (NLS) of NF-kB proteins and keeping them sequestered in an inactive state in the cytoplasm. Also, NFKBIA blocks the ability of NF-κB transcription factors to bind to DNA, which is required for NF-kB's proper functioning. Signal-induced degradation of I kappa B alpha exposes the nuclear localization signal of NF-kappa B, thus allowing it to translocate into the nucleus and activate transcription from responsive genes. An autoregulatory loop is established when NF-kappa B induces expression of the I kappa B alpha gene and newly synthesized I kappa B alpha accumulates in the nucleus where it negatively regulates NF-kappa B-dependent transcription. As part of this post-induction repression, the nuclear export signal on I kappa B alpha mediates the transport of NF-kappa B-I kappa B alpha complexes from the nucleus to the cytoplasm. Deletion of NFKBIA has an effect that is similar to the effect of EGFR amplification in the pathogenesis of glioblastoma and is associated with comparatively short survival. Polymorphisms in NFKBIA may be important in pre-disposition to and outcome after treatment, of multiple myeloma (MM). The NFKBIA gene product, IkappaBalpha, binds to NF-kappaB preventing its activation and is important in mediating resistance to apoptosis in B-cell lymphoproliferative diseases.

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