

## Recombinant Human IKB alpha/NFKBIA Protein (His Tag)

Catalog Number: PKSH030926

**Note:** Centrifuge before opening to ensure complete recovery of vial contents.

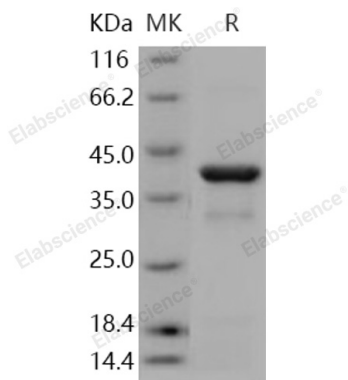
### Description

<b>Species</b>	Human
<b>Source</b>	E.coli-derived Human IKB alpha/NFKBIA protein Phe 2-Leu 317, with an N-terminal His
<b>Calculated MW</b>	36.4 kDa
<b>Observed MW</b>	38 kDa
<b>Accession</b>	NP_065390.1
<b>Bio-activity</b>	Not validated for activity

### Properties

<b>Purity</b>	> 90 % as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	Please contact us for more information.
<b>Storage</b>	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
<b>Shipping</b>	This product is provided as lyophilized powder which is shipped with ice packs.
<b>Formulation</b>	Lyophilized from sterile 50mM Tris, 0.5M NaCl, pH 8.0 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
<b>Reconstitution</b>	Please refer to the printed manual for detailed information.

### Data



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### Background

#### For Research Use Only

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Nuclear factor of kappa light polypeptide gene enhancer in B-cells inhibitor, alpha (I $\kappa$ B 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. In addition, NFKBIA blocks the ability of NF- $\kappa$ B 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 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, I $\kappa$ Balpha, binds to NF-kappaB preventing its activation and is important in mediating resistance to apoptosis in B-cell lymphoproliferative diseases.

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