Phospho-IKB alpha (pTyr42) Antibody [clone 32N56] (FY12745)

Catalog No.	Formulation	Size
FY12745	Rabbit IgG in phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol, 0.4-0.5mg/ml BSA	100 ul

Recombinant RABBIT MONOCLONAL

Bulk quote request

Availability	2-3 weeks
Species Reactivity	Human
Format	Liquid
Host	Rabbit
Clonality	Recombinant Rabbit Monoclonal
Isotype	Rabbit IgG
Clone Name	32N56
Purity	Affinity-chromatography
Buffer	Rabbit IgG in phosphate buffered saline, pH 7.4, 150mM NaCl, 0.02% sodium azide and 50% glycerol, 0.4-0.5mg/ml BSA.
UniProt	P25963
Applications	Western Blot : 1:500-1:2000
Limitations	This Phospho-IKB alpha (pTyr42) antibody is available for research use only.

Description

Phospho-IKB alpha (pTyr42) antibody detects inhibitor of kappa B alpha when phosphorylated at tyrosine 42. This protein, encoded by the NFKBIA gene, is also referred to as NF kappa B inhibitor alpha, MAD3, and IKBA. IKB alpha is a member of the I kappa B protein family, which functions as central regulators of NF kappa B transcription factors. In resting cells, IKB alpha binds NF kappa B dimers such as p65 and p50, sequestering them in the cytoplasm and preventing transcription of inflammatory and survival genes. Phosphorylation at Tyr42 represents a non canonical regulatory event that differs from the classical serine phosphorylation that targets IKB alpha for degradation.

Phospho-IKB alpha (pTyr42) antibody is applied widely in immunology, cancer biology, and signal transduction research. NF kappa B signaling is one of the most important regulators of immunity, inflammation, and cell survival. Detecting Tyr42 phosphorylation allows researchers to distinguish oxidative stress induced NF kappa B activation from canonical TNF and IL1 mediated activation. This distinction is critical for studies dissecting how different stimuli converge on the NF kappa B

pathway.

Phosphorylation at Tyr42 is mediated by kinases such as c Src, Syk, and other non receptor tyrosine kinases. Unlike serine phosphorylation at Ser32 and Ser36, which leads to ubiquitination and degradation of IKB alpha, phosphorylation at Tyr42 can stabilize IKB alpha or change its interactions with NF kappa B subunits. This dual regulatory system provides cells with flexibility to tailor transcriptional responses under stress, infection, or DNA damage. Phospho-IKB alpha (Tyr42) antibody enables researchers to track these unique signaling events.

Applications of this antibody include western blotting, immunohistochemistry, and immunofluorescence. Western blot assays distinguish phosphorylated IKB alpha bands from non modified protein, providing direct evidence of signaling activation. Immunohistochemistry reveals spatial distribution of Tyr42 phosphorylation in tissues such as inflamed mucosa or tumor biopsies. Immunofluorescence detects dynamic changes in subcellular localization, showing how NF kappa B and IKB alpha shuttle between cytoplasm and nucleus after stimulation. These techniques allow mechanistic dissection of cell signaling in both cultured cells and patient samples.

The biological consequences of Tyr42 phosphorylation are complex. In some contexts, phosphorylation promotes apoptosis by releasing NF kappa B from IKB alpha, while in others it stabilizes IKB alpha and suppresses NF kappa B activity. This duality underscores the importance of site specific phospho antibodies. By applying Phospho-IKB alpha (Tyr42) antibody, researchers can resolve conflicting mechanisms and generate precise models of NF kappa B regulation.

Aberrant NF kappa B signaling contributes to oncogenesis, autoimmune disease, and chronic inflammatory disorders. Phosphorylation at Tyr42 has been detected in leukemias, lymphomas, and solid tumors, where altered IKB alpha activity influences tumor cell survival and resistance to therapy. Monitoring this modification with antibody based assays can provide biomarkers for patient stratification and therapeutic development.

In addition to cancer, Tyr42 phosphorylation has been linked to oxidative stress responses in cardiovascular disease, neurodegeneration, and aging. For example, ischemic heart tissue shows altered IKB alpha phosphorylation patterns, while neurons under oxidative stress exhibit abnormal NF kappa B signaling that contributes to cell death. Phospho-IKB alpha (pTyr42) antibody therefore has applications extending beyond immunology into metabolic and neurological research.

Phospho-IKB alpha (pTyr42) antibody from NSJ Bioreagents offers strong specificity and reproducibility. Its validated performance across experimental platforms ensures reliable detection of this phosphorylation event, making it an essential tool for dissecting NF kappa B signaling in health and disease.

Application Notes

Optimal dilution of the Phospho-IKB alpha (pTyr42) antibody should be determined by the researcher.

Immunogen

A synthesized peptide derived from human Phospho-IKB alpha (Y42) was used as the immunogen for the Phospho-IKB alpha (pTyr42) antibody.

Storage

Store the Phospho-IKB alpha (pTyr42) antibody at -20oC.