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Tel: 858.875.1900 Fax: 858.875.1999

Anti-NFkB p100/p52 Antibody

Catalog # ABO11250

Specification

Anti-NFkB p100/p52 Antibody - Product Information

Application	**IHC, WB**
Primary Accession	Q9WTK5
Host	**Rabbit**
Reactivity	**Mouse, Rat**
Clonality	**Polyclonal**
Format	**Lyophilized**

Description
Rabbit IgG polyclonal antibody for Nuclear factor NF-kappa-B p100 subunit(NFKB2) detection. Tested with WB, IHC-P in Mouse;Rat.

Reconstitution
Add 0.2ml of distilled water will yield a concentration of 500ug/ml.

Anti-NFkB p100/p52 Antibody - Additional Information

Gene ID 18034

Other Names
Nuclear factor NF-kappa-B p100 subunit, DNA-binding factor KBF2, Nuclear factor of kappa light polypeptide gene enhancer in B-cells 2, Nuclear factor NF-kappa-B p52 subunit, Nfkb2

Calculated MW
96832 MW KDa

Application Details
Immunohistochemistry(Paraffin-embedded Section), 0.5-1 µg/ml, Rat, Mouse, By Heat
Western blot, 0.1-0.5 µg/ml, Mouse, Rat

Subcellular Localization
Nucleus . Cytoplasm . Nuclear, but also found in the cytoplasm in an inactive form complexed to an inhibitor (I-kappa-B). .

Tissue Specificity
Highly expressed in lymph nodes and thymus. .

Protein Name
Nuclear factor NF-kappa-B p100 subunit

Contents
Each vial contains 5mg BSA, 0.9mg NaCl, 0.2mg Na2HPO4, 0.05mg Thimerosal, 0.05mg NaN3.

Immunogen
A synthetic peptide corresponding to a sequence at the N-terminus of mouse NFkB p100(5-21aa YDPGLDGIPEYDDFEFS), identical to the related rat sequence.

Purification
Immunogen affinity purified.

Cross Reactivity
No cross reactivity with other proteins

Storage

At -20˚C for one year. After r˚Constitution, at 4˚C for one month. It˚Can also be aliquotted and stored frozen at -20˚C for a longer time.Avoid repeated freezing and thawing.

Sequence Similarities
Contains 7 ANK repeats.

Anti-NFkB p100/p52 Antibody - Protein Information

Name Nfkb2

Function
NF-kappa-B is a pleiotropic transcription factor present in almost all cell types and is the endpoint of a series of signal transduction events that are initiated by a vast array of stimuli related to many biological processes such as inflammation, immunity, differentiation, cell growth, tumorigenesis and apoptosis. NF-kappa-B is a homo- or heterodimeric complex formed by the Rel-like domain- containing proteins RELA/p65, RELB, NFKB1/p105, NFKB1/p50, REL and NFKB2/p52. The dimers bind at kappa-B sites in the DNA of their target genes and the individual dimers have distinct preferences for different kappa-B sites that they can bind with distinguishable affinity and specificity. Different dimer combinations act as transcriptional activators or repressors, respectively. NF-kappa-B is controlled by various mechanisms of post-translational modification and subcellular compartmentalization as well as by interactions with other cofactors or corepressors. NF-kappa-B complexes are held in the cytoplasm in an inactive state complexed with members of the NF-kappa-B inhibitor (I- kappa-B) family. In a conventional activation pathway, I-kappa-B is phosphorylated by I-kappa-B kinases (IKKs) in response to different activators, subsequently degraded thus liberating the active NF-kappa-B complex which translocates to the nucleus. In a non-canonical activation pathway, the MAP3K14-activated CHUK/IKKA homodimer phosphorylates NFKB2/p100 associated with RelB, inducing its proteolytic processing to NFKB2/p52 and the formation of NF-kappa-B RelB-p52 complexes. The NF-kappa-B heterodimeric RelB-p52 complex is a transcriptional activator. The NF-kappa-B p52-p52 homodimer is a transcriptional repressor. NFKB2 appears to have dual functions such as cytoplasmic retention of attached NF-kappa-B proteins by p100 and generation of p52 by a cotranslational processing. The proteasome- mediated process ensures the production of both p52 and p100 and preserves their independent function. p52 binds to the kappa-B consensus sequence 5'-GGRNNYYCC-3', located in the enhancer region of genes involved in immune response and acute phase reactions. p52 and p100 are respectively the minor and major form; the processing of p100 being relatively poor. Isoform p49 is a subunit of the NF-kappa-B protein complex, which stimulates the HIV enhancer in synergy with p65 (By similarity). In concert with RELB, regulates the circadian clock by repressing the transcriptional activator activity of the CLOCK-BMAL1 heterodimer.

Cellular Location
Nucleus. Cytoplasm. Note=Nuclear, but also found in the cytoplasm in an inactive form complexed to an inhibitor (I-kappa-B).

Tissue Location
Highly expressed in lymph nodes and thymus.

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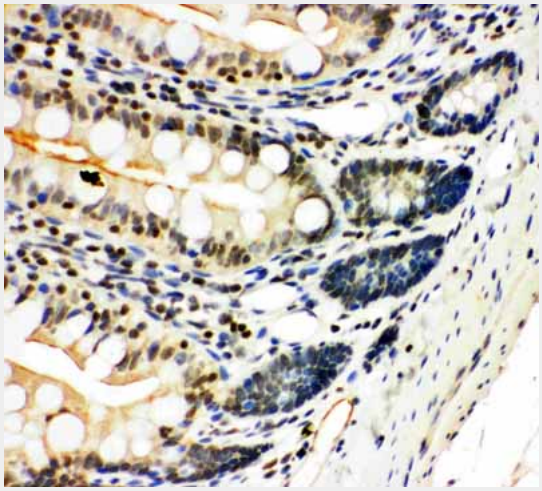
Anti-NFkB p100/p52 Antibody - Protocols

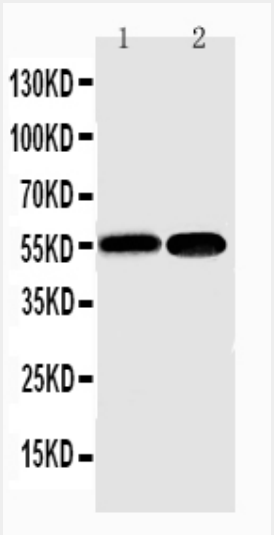
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Anti-NFkB p100/p52 Antibody - Images



Anti-NFkB p100/p52 antibody, ABO11250, IHC(P)IHC(P): Rat Intestine Tissue



Anti-NFkB p100/p52 antibody, ABO11250, Western blottingLane 1: Mouse Liver Tissue LysateLane 2: HEPA Cell Lysate

Anti-NFkB p100/p52 Antibody - Background

NFKB2(Nuclear Factor Kappa-B, Subunit 2), also known as NFKB or p52/p100 SUBUNIT, is a protein that in humans is encoded by the NFKB2 gene. Liptay et al.(1992) mapped the gene for what they

called the p49/p100 subunit of NFKB(NFKB2) to chromosome 10 by Southern blot analysis of panels of human/Chinese hamster cell hybrids. By fluorescence in situ hybridization(FISH), they confirmed the localization and mapped the gene with greater resolution to 10q24. NFKB2 appears to be the same as LYT10. Claudio et al.(2002) showed that bone marrow(BM) cells from Nfkb2-deficient mice, but not Nfkb1-deficient mice, failed to increase relative and total IgD-positive transitional-1(T1) stage B cells in response to Baff. In vivo, however, Nfkb2-deficient mice did generate mature B cells, but at reduced numbers. Mice of the aly/aly strain, which are naturally deficient in Nik, and mice of the A/WySNJ strain, which have a mutation in Baffr, also failed to produce T1 B cells in response to Baff. Baff stimulation enhanced expression of Bcl2 in T1 B cells, thereby promoting B-cell survival, and caused the processing of the p100 form of Nfkb2 to p52, which again required Baffr and Nik, but not Nemo(IKKG). Immunoblot analysis showed that BM cells contained primarily p100.