

## Product datasheet for **TA319202**

### **NF-kB p65 (RELA) Rabbit Polyclonal Antibody**

#### **Product data:**

Product Type:	Primary Antibodies
Applications:	IHC, WB
Recommended Dilution:	ELISA: 1:1,000 - 1:6,000, WB: 1:200 - 1:2,000, IHC: 5 ug/ml
Reactivity:	Human
Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Immunogen:	This affinity purified antibody was prepared from whole rabbit serum produced by repeated immunizations with a synthetic peptide corresponding to residues surrounding S536 of human p65 (RelA) protein.
Formulation:	0.02 M Potassium Phosphate, 0.15 M Sodium Chloride, pH 7.2
Concentration:	lot specific
Conjugation:	Unconjugated
Storage:	Store at -20°C as received.
Stability:	Stable for 12 months from date of receipt.
Gene Name:	RELA proto-oncogene, NF-kB subunit
Database Link:	<a href="#">NP_001138610</a> <a href="#">Entrez Gene 5970 Human</a> <a href="#">Q04206</a>
Synonyms:	NFKB3; p65



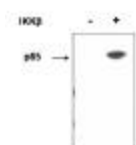
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**Note:** This antibody is suitable for Cancer, Immunology and Nuclear Signaling research. NFkB was originally identified as a factor that binds to the immunoglobulin kappa light chain enhancer in B cells. It was subsequently found in non-B cells in an inactive cytoplasmic form consisting of NFkB bound to IκB. NFkB was originally identified as a heterodimeric DNA binding protein complex consisting of p65 (RelA) and p50 (NFkB1) subunits. Other identified subunits include p52 (NFkB2), cRel, and RelB. The p65, cRel, and RelB subunits are responsible for transactivation. The p50 and p52 subunits possess DNA binding activity but limited ability to transactivate. p52 has been reported to form transcriptionally active heterodimers with the NFkB subunit p65, similar to p50/p65 heterodimers. Low levels of p52 and p50 homodimers can also exist in cells. The heterodimers of p52/p65 and p50/p65 are regulated by physical inactivation in the cytoplasm by IκB-α. IκB-α binds to the p65 subunit, preventing nuclear localization, and DNA binding. Activators mediate a rapid phosphorylation of IκB by IκB kinase (IKK), which results in subsequent ubiquitination and proteolytic degradation. NFkB is then transported to the nucleus, where it activates transcription of target genes through binding to NFkB target sequences within the promoter. The HTLV-I protein Tax can induce constitutive NFkB activation through phosphorylation of both IκB-α and IκB-β. The transforming protein Tax inhibits p53 transcriptional activity through the NFkB signaling pathway, specifically via the p65 (RelA) subunit. The inhibition of p53 activity is dependent upon phosphorylation of p65 (RelA) at S536 by the upstream kinase IKKβ.

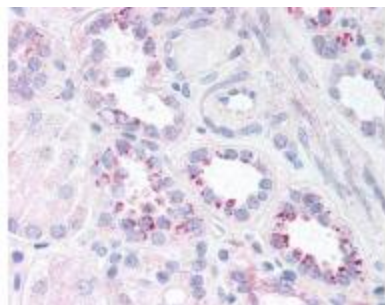
**Protein Families:** Druggable Genome, Transcription Factors

**Protein Pathways:** Acute myeloid leukemia, Adipocytokine signaling pathway, Apoptosis, B cell receptor signaling pathway, Chemokine signaling pathway, Chronic myeloid leukemia, Cytosolic DNA-sensing pathway, Epithelial cell signaling in Helicobacter pylori infection, MAPK signaling pathway, Neurotrophin signaling pathway, NOD-like receptor signaling pathway, Pancreatic cancer, Pathways in cancer, Prostate cancer, RIG-I-like receptor signaling pathway, Small cell lung cancer, T cell receptor signaling pathway, Toll-like receptor signaling pathway

## Product images:



Western blot using affinity purified anti-p65 (RelA) pS536 antibody shows detection of p65 phosphorylated at S536. The control blot (left lane) contains 100 ng of purified p65-GST fusion protein. The band is seen (right lane) when this protein is phosphorylated at S536 by IKKβ. Personal Communication. J. Brady, NCI, Bethesda, MD.



Anti-p65 (RelA) pS536 antibody was used at 5.0 µg/ml to detect signal in a variety of tissues including multi-human, multi-brain and multi-cancer slides. This image shows moderate positive staining of human kidney distal tubules and collecting ducts. Tissue was formalin-fixed and paraffin embedded. The image shows localization of the antibody as the precipitated red signal, with a hematoxylin purple nuclear counterstain. Personal Communication, Tina Roush, LifeSpanBiosciences, Seattle, WA.