

# Product datasheet for TA319204

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

### **Product data:**

#### OriGene Technologies, Inc.

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Product Type:	Primary Antibodies
Applications:	WB
Recommended Dilution:	ELISA: 1:5,000 - 1:25,000, WB: 1:1,000
Reactivity:	Human
Host:	Rabbit
lsotype:	IgG
Clonality:	Polyclonal
Immunogen:	NFkB p65 (Rel A) peptide corresponding to a region near phospho Serine 276 of the human protein conjugated to Keyhole Limpet Hemocyanin (KLH).
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:	<u>NP_001138610</u> <u>Entrez Gene 5970 Human</u> <u>Q04206</u>
Synonyms:	NFKB3; p65



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#### **GRIGENE** NF-kB p65 (RELA) Rabbit Polyclonal Antibody – TA319204

NF?B was originally identified as a factor that binds to the immunoglobulin kappa light chain Note: enhancer in B cells. It was subsequently found in non-B cells in an inactive cytoplasmic form consisting of NF?B bound to I?B. NF?B 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 NF?B 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 I?B-a. I?B-a ?binds to the p65 subunit preventing nuclear localization and DNA binding. Activators mediate a rapid phosphorylation of I?B by I?B kinase (IKK) which results in subsequent ubiquitination and proteolytic degradation. NF?B is then transported to the nucleus, where it activates transcription of target genes through binding to NF?B target sequences within the promoter. The HTLV-I protein Tax can induce constitutive NF?B activation through phosphorylation of both I?B-a and I?B-β. The transforming protein Tax inhibits p53 transcriptional activity through the NF?B 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<br/>pathway, Chemokine signaling pathway, Chronic myeloid leukemia, Cytosolic DNA-sensing<br/>pathway, Epithelial cell signaling in Helicobacter pylori infection, MAPK signaling pathway,<br/>Neurotrophin signaling pathway, NOD-like receptor signaling pathway, Pancreatic cancer,<br/>Pathways in cancer, Prostate cancer, RIG-I-like receptor signaling pathway, Small cell lung<br/>cancer, T cell receptor signaling pathway, Toll-like receptor signaling pathway

### **Product images:**



Affinity purified Anti-NFKB p65 (Rel A) pS276 TA319204 lot 24040 was probed against Normal (Lane 1) and TNFalpha (Lane 2) Stimulated HeLa whole cell lysates. A band was observed between 55 and 72 kD corresponding to the expected MW of NFkB p65. The observed higher MW bands have not been characterized.

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