# Anti-NAK / TBK1 Antibody [2G1]

## RT1413

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| **Product Type:** | Mouse monoclonal IgG1, primary antibodies |
| **Species reactivity:** | Human, Mouse, Rat |
| **Applications:** | WB, IP |
| **Molecular Wt:** | 80 kDa |
| **Clone number:** | 2G1 |

**Description:** The transcription factor NFκB is retained in the cytoplasm in an inactive form by the inhibitory protein IκB. Activation of NFκB requires that IκB be phosphorylated on specific serine residues, which results in targeted degradation of IκB. IκB kinase α (IKKα), previously designated CHUK, interacts with IκB-α and specifically phosphorylates IκB-α on the sites that trigger its degradation, Serines 32 and 36. The functional IKK complex contains three subunits, IKKα, IKKβ and IKKγ (also designated NEMO), and each appear to make essential contributions to IκB phosphorylation. TANK binding kinase (TBK1), also designated T2K, is a novel IKK-related kinase that has been identified in murine and human tissues. TBK1 was shown to complex with TRAF2 and TANK in the NFκB activation pathway. TBK1 shares homology with IKKα and IKKβ in the amino-terminal half, which includes the kinase domain.

**Immunogen:** Synthetic peptide within Human TBK1 aa 563-577.

**Positive control:** RAW264.7, MCF7, KNRK.

**Subcellular location:** Cytoplasm.

**Database links:** SwissProt: Q9UHD2 Human

**Recommended Dilutions:**

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| **WB** | 1:100-1:1,000 |
| **IP** | 1-2 μg per 100-500 μg of total protein(1 ml of cell lysate) |

**Storage Buffer:** 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

**Storage Instruction:** Store at +4℃

**Purity:** Protein A affinity purified.

## Images

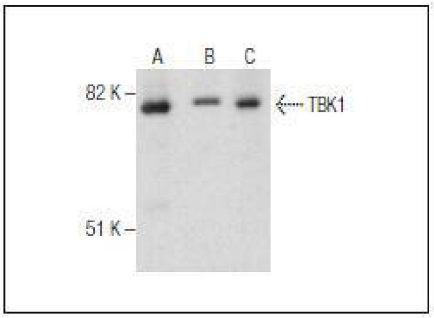


**Fig1:** Western blot analysis of TBK1 expression in RAW264.7 (A), MCF7 (B) and KNRK (C) whole cell lysates.



## Background References

1. Chen, H., et al. 2011. Activation of STAT6 by STING is critical for antiviral innate immunity. Cell 147: 436-446.
2. Neerincx, A., et al. 2010. A role for the human NLR family member NLRC5 in antiviral responses. J. Biol. Chem. 285: 26223-26232.