

**TRIB3 Antibody (C-term) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP8157b****Specification**

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**TRIB3 Antibody (C-term) Blocking peptide - Product Information**Primary Accession [Q96RU7](#)**TRIB3 Antibody (C-term) Blocking peptide - Additional Information****Gene ID** 57761**Other Names**

Tribbles homolog 3, TRB-3, Neuronal cell death-inducible putative kinase, SINK, p65-interacting inhibitor of NF-kappa-B, TRIB3, C20orf97, NIPK, SKIP3, TRB3

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP8157b](/product/products/AP8157b) was selected from the C-term region of human NPK . A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**TRIB3 Antibody (C-term) Blocking peptide - Protein Information****Name** TRIB3**Synonyms** C20orf97, NIPK, SKIP3, TRB3**Function**

Inactive protein kinase which acts as a regulator of the integrated stress response (ISR), a process for adaptation to various stress (PubMed: [15775988](http://www.uniprot.org/citations/15775988), PubMed: [15781252](http://www.uniprot.org/citations/15781252)). Inhibits the transcriptional activity of DDIT3/CHOP and is involved in DDIT3/CHOP-dependent cell death during ER stress (PubMed: [15775988](http://www.uniprot.org/citations/15775988), PubMed: [15781252](http://www.uniprot.org/citations/15781252)). May play a role in programmed neuronal cell death but does not appear to affect non-neuronal cells (PubMed: [15775988](http://www.uniprot.org/citations/15775988),

PubMed:<a href="http://www.uniprot.org/citations/15781252" target="\_blank">15781252</a>). Acts as a negative feedback regulator of the ATF4-dependent transcription during the ISR: while TRIB3 expression is promoted by ATF4, TRIB3 protein interacts with ATF4 and inhibits ATF4 transcription activity (By similarity). Disrupts insulin signaling by binding directly to Akt kinases and blocking their activation (By similarity). May bind directly to and mask the 'Thr-308' phosphorylation site in AKT1 (By similarity). Interacts with the NF-kappa-B transactivator p65 RELA and inhibits its phosphorylation and thus its transcriptional activation activity (PubMed:<a href="http://www.uniprot.org/citations/12736262" target="\_blank">12736262</a>). Interacts with MAPK kinases and regulates activation of MAP kinases (PubMed:<a href="http://www.uniprot.org/citations/15299019" target="\_blank">15299019</a>). Can inhibit APOBEC3A editing of nuclear DNA (PubMed:<a href="http://www.uniprot.org/citations/22977230" target="\_blank">22977230</a>).

### **Cellular Location**

Nucleus.

### **Tissue Location**

Highest expression in liver, pancreas, peripheral blood leukocytes and bone marrow. Also highly expressed in a number of primary lung, colon and breast tumors. Expressed in spleen, thymus, and prostate and is undetectable in other examined tissues, including testis, ovary, small intestine, colon, leukocyte, heart, brain, placenta, lung, skeletal muscle, and kidney

### **TRIB3 Antibody (C-term) Blocking peptide - Protocols**

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

- [Blocking Peptides](#)

### **TRIB3 Antibody (C-term) Blocking peptide - Images**

### **TRIB3 Antibody (C-term) Blocking peptide - Background**

NPK is a putative protein kinase that is induced by the transcription factor NF-kappaB. The encoded protein is a negative regulator of NF-kappaB and can also sensitize cells to TNF- and TRAIL-induced apoptosis. In addition, this protein can negatively regulate the cell survival serine-threonine kinase AKT1.

### **TRIB3 Antibody (C-term) Blocking peptide - References**

Wu, M., et al., J. Biol. Chem. 278(29):27072-27079 (2003). Du, K., et al., Science 300(5625):1574-1577 (2003). Bowers, A.J., et al., Oncogene 22(18):2823-2835 (2003).