

# **TOLLIP Antibody (C-term) Blocking Peptide**

Synthetic peptide Catalog # BP2163b

## **Specification**

# **TOLLIP Antibody (C-term) Blocking Peptide - Product Information**

Primary Accession

**Q9H0E2** 

# **TOLLIP Antibody (C-term) Blocking Peptide - Additional Information**

**Gene ID 54472** 

#### **Other Names**

Toll-interacting protein, TOLLIP

## Target/Specificity

The synthetic peptide sequence used to generate the antibody <a href=/product/products/AP2163b>AP2163b</a> was selected from the C-term region of human TOLLIP . 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.

# **TOLLIP Antibody (C-term) Blocking Peptide - Protein Information**

# **Name TOLLIP**

### **Function**

Component of the signaling pathway of IL-1 and Toll-like receptors (PubMed:<a href="http://www.uniprot.org/citations/10854325" target="\_blank">10854325</a>, PubMed:<a href="http://www.uniprot.org/citations/11751856" target="\_blank">11751856</a>). Inhibits cell activation by microbial products. Recruits IRAK1 to the IL-1 receptor complex (PubMed:<a href="http://www.uniprot.org/citations/10854325" target="\_blank">10854325</a>). Inhibits IRAK1 phosphorylation and kinase activity (PubMed:<a href="http://www.uniprot.org/citations/11751856" target="\_blank">11751856</a>). Connects the

href="http://www.uniprot.org/citations/11751856" target="\_blank">11751856</a>). Connects the ubiquitin pathway to autophagy by functioning as a ubiquitin-ATG8 family adapter and thus mediating autophagic clearance of ubiquitin conjugates (PubMed:<a

 $href="http://www.uniprot.org/citations/25042851" target="\_blank">25042851</a>). The TOLLIP-dependent selective autophagy pathway plays an important role in clearance of cytotoxic polyQ proteins aggregates (PubMed:<a href="http://www.uniprot.org/citations/25042851" target="_blank">25042851</a>). The$ 



target="\_blank">25042851</a>). In a complex with TOM1, recruits ubiquitin-conjugated proteins onto early endosomes (PubMed:<a href="http://www.uniprot.org/citations/15047686" target="\_blank">15047686</a>). Binds to phosphatidylinositol 3-phosphate (PtdIns(3)P) (PubMed:<a href="http://www.uniprot.org/citations/26320582" target=" blank">26320582</a>).

### **Cellular Location**

Cytoplasm. Endosome. Early endosome Note=Localized to endo/exosomal vesicles

### **TOLLIP Antibody (C-term) Blocking Peptide - Protocols**

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

### Blocking Peptides

### **TOLLIP Antibody (C-term) Blocking Peptide - Images**

# **TOLLIP Antibody (C-term) Blocking Peptide - Background**

Toll like protein is a component of the signaling pathway of IL1 and Toll like receptors. It inhibits cell activation by microbial products. Tollip recruits IRAK1 to the IL1 receptor complex and inhibits IRAK1 phosphorylation and kinase activity. It oligomerizes and binds to TLR2 and the TLR4-MD2 complex via its C terminus. It exists as a complex with IRAK1 in unstimulated cells. Upon IL1 signaling, Tollip binds to the activated IL1 receptor complex containing IL-1RI, IL-1RacP and the adapter protein MyD88, where it interacts with the TIR domain of IL-1RacP. MyD88 then triggers IRAK1 autophosphorylation, which in turn leads to the dissociation of IRAK1 from Tollip and IL-1RAcP. TOLLIP also interacts with TLR2 and TLR4; TOLLIP overexpression inhibits nuclear factor kappa-B (NFKB) activation in response to lipopolysaccharide and IL1B.

## **TOLLIP Antibody (C-term) Blocking Peptide - References**

Zhang, G., et al., J. Biol. Chem. 277(9):7059-7065 (2002).Bulut, Y., et al., J. Immunol. 167(2):987-994 (2001).Burns, K., et al., Nat. Cell Biol. 2(6):346-351 (2000).Volpe, F., et al., FEBS Lett. 419(1):41-44 (1997).