

**TLR2 Antibody (Center) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP13694c****Specification**

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**TLR2 Antibody (Center) Blocking peptide - Product Information**Primary Accession [O60603](#)**TLR2 Antibody (Center) Blocking peptide - Additional Information****Gene ID** 7097**Other Names**

Toll-like receptor 2, Toll/interleukin-1 receptor-like protein 4, CD282, TLR2, TIL4

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody AP13694c was selected from the Center region of TLR2. 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.

**TLR2 Antibody (Center) Blocking peptide - Protein Information****Name** TLR2 ([HGNC:11848](#))**Synonyms** TIL4**Function**

Cooperates with LY96 to mediate the innate immune response to bacterial lipoproteins and other microbial cell wall components. Cooperates with TLR1 or TLR6 to mediate the innate immune response to bacterial lipoproteins or lipopeptides (PubMed:<a href="http://www.uniprot.org/citations/17889651" target="\_blank">17889651</a>, PubMed:<a href="http://www.uniprot.org/citations/21078852" target="\_blank">21078852</a>). Acts via MYD88 and TRAF6, leading to NF-kappa-B activation, cytokine secretion and the inflammatory response. May also activate immune cells and promote apoptosis in response to the lipid moiety of lipoproteins (PubMed:<a href="http://www.uniprot.org/citations/10426995" target="\_blank">10426995</a>, PubMed:<a href="http://www.uniprot.org/citations/10426996" target="\_blank">10426996</a>). Recognizes mycoplasmal macrophage-activating lipopeptide-2kD (MALP-2), soluble tuberculosis factor (STF), phenol-soluble modulin (PSM) and

B.burgdorferi outer surface protein A lipoprotein (OspA-L) cooperatively with TLR6 (PubMed:<a href="http://www.uniprot.org/citations/11441107" target="\_blank">11441107</a>). Stimulation of monocytes in vitro with M.tuberculosis PstS1 induces p38 MAPK and ERK1/2 activation primarily via this receptor, but also partially via TLR4 (PubMed:<a href="http://www.uniprot.org/citations/16622205" target="\_blank">16622205</a>). MAPK activation in response to bacterial peptidoglycan also occurs via this receptor (PubMed:<a href="http://www.uniprot.org/citations/16622205" target="\_blank">16622205</a>). Acts as a receptor for M.tuberculosis lipoproteins LprA, LprG, LpqH and PstS1, some lipoproteins are dependent on other coreceptors (TLR1, CD14 and/or CD36); the lipoproteins act as agonists to modulate antigen presenting cell functions in response to the pathogen (PubMed:<a href="http://www.uniprot.org/citations/19362712" target="\_blank">19362712</a>). M.tuberculosis HSP70 (dnaK) but not HSP65 (groEL-2) acts via this protein to stimulate NF-kappa-B expression (PubMed:<a href="http://www.uniprot.org/citations/15809303" target="\_blank">15809303</a>). Recognizes M.tuberculosis major T-antigen EsxA (ESAT-6) which inhibits downstream MYD88-dependent signaling (shown in mouse) (By similarity). Forms activation clusters composed of several receptors depending on the ligand, these clusters trigger signaling from the cell surface and subsequently are targeted to the Golgi in a lipid-raft dependent pathway. Forms the cluster TLR2:TLR6:CD14:CD36 in response to diacylated lipopeptides and TLR2:TLR1:CD14 in response to triacylated lipopeptides (PubMed:<a href="http://www.uniprot.org/citations/16880211" target="\_blank">16880211</a>). Required for normal uptake of M.tuberculosis, a process that is inhibited by M.tuberculosis LppM (By similarity).

#### **Cellular Location**

Membrane {ECO:0000250|UniProtKB:Q9QUN7}; Single- pass type I membrane protein. Cytoplasmic vesicle, phagosome membrane {ECO:0000250|UniProtKB:Q9QUN7}; Single-pass type I membrane protein. Membrane raft. Note=Does not reside in lipid rafts before stimulation but accumulates increasingly in the raft upon the presence of the microbial ligand. In response to diacylated lipoproteins, TLR2:TLR6 heterodimers are recruited in lipid rafts, this recruitment determines the intracellular targeting to the Golgi apparatus. Triacylated lipoproteins induce the same mechanism for TLR2:TLR1 heterodimers.

#### **Tissue Location**

Highly expressed in peripheral blood leukocytes, in particular in monocytes, in bone marrow, lymph node and in spleen. Also detected in lung and in fetal liver. Levels are low in other tissues

### **TLR2 Antibody (Center) Blocking peptide - Protocols**

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

- [Blocking Peptides](#)

### **TLR2 Antibody (Center) Blocking peptide - Images**

### **TLR2 Antibody (Center) Blocking peptide - Background**

The protein encoded by this gene is a member of the Toll-like receptor (TLR) family which plays a fundamental role in pathogen recognition and activation of innate immunity. TLRs are highly conserved from Drosophila to humans and share structural and functional similarities. They recognize pathogen-associated molecular patterns (PAMPs) that are expressed on infectious agents, and mediate the production of cytokines necessary for the development of effective immunity. The various TLRs exhibit different patterns of expression. This gene is expressed most abundantly in peripheral blood leukocytes, and mediates host response to Gram-positive bacteria and yeast via stimulation of NF-kappaB.

### **TLR2 Antibody (Center) Blocking peptide - References**

Chambers, M.A., et al. Biochem. Biophys. Res. Commun. 400(3):403-408(2010)Franchim, C.S., et al. Hypertens Pregnancy (2010) In press :Selvaraj, P., et al. Tuberculosis (Edinb) 90(5):306-310(2010)Xie, F., et al. Hypertens Pregnancy 29(4):390-398(2010)Qian, F.H., et al. J Investig Allergol Clin Immunol 20(4):340-346(2010)