

M TLR4 Antibody (N-term) Blocking peptide
Synthetic peptide
Catalog # BP1504a**Specification**

M TLR4 Antibody (N-term) Blocking peptide - Product InformationPrimary Accession [Q9OUK6](#)**M TLR4 Antibody (N-term) Blocking peptide - Additional Information****Gene ID** 21898**Other Names**

Toll-like receptor 4, CD284, Tlr4, Lps

Target/Specificity

The synthetic peptide sequence is selected from aa 39~55 of mouse TLR4.

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.

M TLR4 Antibody (N-term) Blocking peptide - Protein Information**Name** Tlr4**Synonyms** Lps**Function**

Transmembrane receptor that functions as a pattern recognition receptor recognizing pathogen- and damage-associated molecular patterns (PAMPs and DAMPs) to induce innate immune responses via downstream signaling pathways (PubMed:9851930, PubMed:9989976, PubMed:20133493). At the plasma membrane, cooperates with LY96 to mediate the innate immune response to bacterial lipopolysaccharide (LPS) (PubMed:9851930, PubMed:9989976, PubMed:20133493). Also involved in LPS-independent inflammatory responses triggered by free fatty acids, such as palmitate, and Ni(2+). Mechanistically, acts via MYD88, TIRAP and TRAF6, leading to NF-kappa-B activation, cytokine secretion and the inflammatory

response (PubMed:24380872). Alternatively, CD14- mediated TLR4 internalization via endocytosis is associated with the initiation of a MYD88-independent signaling via the TICAM1-TBK1-IRF3 axis leading to type I interferon production. In addition to the secretion of proinflammatory cytokines, initiates the activation of NLRP3 inflammasome and formation of a positive feedback loop between autophagy and NF-kappa-B signaling cascade. In complex with TLR6, promotes inflammation in monocytes/macrophages by associating with TLR6 and the receptor CD86. Upon ligand binding, such as oxLDL or amyloid- beta 42, the TLR4:TLR6 complex is internalized and triggers inflammatory response, leading to NF-kappa-B-dependent production of CXCL1, CXCL2 and CCL9 cytokines, via MYD88 signaling pathway, and CCL5 cytokine, via TICAM1 signaling pathway. In myeloid dendritic cells, vesicular stomatitis virus glycoprotein G but not LPS promotes the activation of IRF7, leading to type I IFN production in a CD14- dependent manner (By similarity).

Cellular Location

Cell membrane; Single-pass type I membrane protein. Early endosome {ECO:0000250|UniProtKB:O00206}. Cell projection, ruffle. Note=Upon complex formation with CD36 and TLR6, internalized through dynamin-dependent endocytosis Colocalizes with RFTN1 at cell membrane and then together with RFTN1 moves to endosomes, upon lipopolysaccharide stimulation {ECO:0000250|UniProtKB:O00206}

Tissue Location

Expressed in macrophages (at protein level) (PubMed:28098138, PubMed:35896747). Highly expressed in heart, spleen, lung and muscle. Lower levels are found in liver and kidney (PubMed:23812099).

M TLR4 Antibody (N-term) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

M TLR4 Antibody (N-term) Blocking peptide - Images

M TLR4 Antibody (N-term) Blocking peptide - Background

TLR4, a type I membrane protein that belongs to the Toll-like receptor family, cooperates with LY96 and CD14 to mediate the innate immune response to bacterial lipopolysaccharide (LPS). It acts via MyD88, TIRAP and TRAF6, leading to NF-kappa-B activation, cytokine secretion and the inflammatory response TLR4 Belongs to the lipopolysaccharide (LPS) receptor, a multi-protein complex containing at least CD14, LY96 and TLR. TLR4 binds to LY96 via the extracellular domain, and to MyD88 and TIRAP via their respective TIR domains. The protein contains 19 leucine-rich (LRR) repeats, and It is highly expressed in heart, spleen, lung and muscle. Lower levels are found in liver and kidney. Interstrain analyses reveal that TLR4 is a polymorphic protein and that the extracellular domain is far more variable than the cytoplasmic domain, which is variable at the C-terminal.

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Okazaki, Y., et al., Nature 420(6915):563-573 (2002).Rhee, S.H., et al., J. Biol. Chem. 275(44):34035-34040 (2000).Qureshi, S.T., et al., J. Exp. Med. 189(4):615-625 (1999).Underhill, D.M., et al., Nature 401(6755):811-815 (1999).Poltorak, A., et al., Blood Cells Mol. Dis. 24(3):340-355 (1998).