

Mouse TLR1 Antibody (N-term) Blocking Peptide Synthetic peptide Catalog # BP1501a

Specification

Mouse TLR1 Antibody (N-term) Blocking Peptide - Product Information

Primary Accession

<u>Q9EPQ1</u>

Mouse TLR1 Antibody (N-term) Blocking Peptide - Additional Information

Gene ID 21897

Other Names Toll-like receptor 1, Toll/interleukin-1 receptor-like protein, TIL, CD281, TIr1

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP1501a was selected from the N-term region of human Mouse TLR1 . 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.

Mouse TLR1 Antibody (N-term) Blocking Peptide - Protein Information

Name Tlr1

Function

Participates in the innate immune response to microbial agents. Specifically recognizes diacylated and triacylated lipopeptides. Cooperates with TLR2 to mediate the innate immune response to bacterial lipoproteins or lipopeptides. Forms the activation cluster TLR2:TLR1:CD14 in response to triacylated lipopeptides, this cluster triggers signaling from the cell surface and subsequently is targeted to the Golgi in a lipid-raft dependent pathway. Acts via MYD88 and TRAF6, leading to NF-kappa-B activation, cytokine secretion and the inflammatory response (By similarity). Acts as a coreceptor for M.tuberculosis lipoproteins LprG, LpqH and PhoS1 (pstS1), in conjunction with TLR2 and for some but not all lipoproteins CD14 and/or CD36. The lipoproteins act as agonists to modulate antigen presenting cell functions in response to the pathogen (PubMed:19362712).

Cellular Location



Cell membrane; Single-pass type I membrane protein. Cytoplasmic vesicle, phagosome membrane; Single-pass type I membrane protein. Membrane raft {ECO:0000250|UniProtKB:Q15399}. Golgi apparatus {ECO:0000250|UniProtKB:Q15399}. 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 triacylated lipoproteins, TLR2:TLR1 heterodimers are recruited in lipid rafts, this recruitment determine the intracellular targeting to the Golgi apparatus. {ECO:0000250|UniProtKB:Q15399}

Mouse TLR1 Antibody (N-term) Blocking Peptide - Protocols

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

<u>Blocking Peptides</u>

Mouse TLR1 Antibody (N-term) Blocking Peptide - Images

Mouse TLR1 Antibody (N-term) Blocking Peptide - Background

Higher animals establish host defense by orchestrating innate and adaptive immunity. This is mediated by professional antigen presenting cells, i.e. dendritic cells (DCs). DCs can incorporate pathogens, produce a variety of cytokines, maturate, and present pathogen-derived peptides to T cells, thereby inducing T cell activation and differentiation. These responses are triggered by microbial recognition through type I transmembrane proteins, Toll-like receptors (TLRs) on DCs. TLRs consist of ten members and each TLR is involved in recognizing a variety of microorganism-derived molecular structures. TLR ligands include cell wall components, proteins, nucleic acids, and synthetic chemical compounds, all of which can activate DCs as immune adjuvants. Each TLR can activate DCs in a similar, but distinct manner. For example, TLRs can be divided into subgroups according to their type I interferon (IFN) inducing ability. TLR2 cannot induce IFN-alpha or IFN-beta, but TLR4 can lead to IFN-beta production. Meanwhile, TLR3, TLR7, and TLR9 can induce both IFN-alpha and IFN-beta. Recent evidences suggest that cytoplamic adapters for TLRs are especially crucial for this functional heterogeneity.

Mouse TLR1 Antibody (N-term) Blocking Peptide - References

Hajjar, A.M., et al., J. Immunol. 166(1):15-19 (2001).Ozinsky, A., et al., Proc. Natl. Acad. Sci. U.S.A. 97(25):13766-13771 (2000).