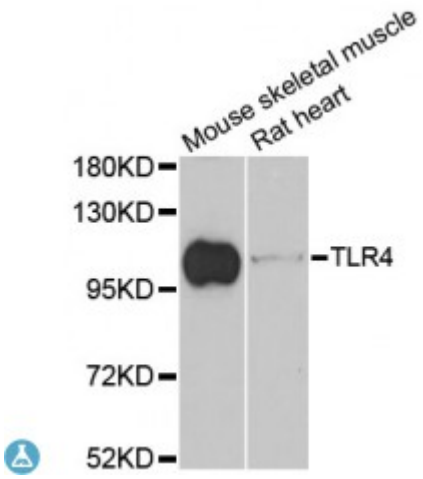
Anti-TLR4 Antibody



Description	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 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 receptor has been implicated in signal transduction events induced by lipopolysaccharide (LPS) found in most gram-negative bacteria. Mutations in this gene have been associated with differences in LPS responsiveness. Multiple transcript variants encoding different isoforms have been found for this gene.

Model	STJ113740
Host	Rabbit
Reactivity	Mouse, Rat
Applications	WB
Immunogen	A synthetic peptide corresponding to a sequence within amino acids 500-600 of human TLR4 (NP_612564.1).
Gene ID	7099
Gene Symbol	TLR4
Dilution range	WB 1:500 - 1:2000
Tissue Specificity	Highly expressed in placenta, spleen and peripheral blood leukocytes
Purification	Affinity purification

Note	For Research Use Only (RUO).
Protein Name	Toll-like receptor 4 hToll CD antigen CD284
Molecular Weight	95.68 kDa
Clonality	Polyclonal
Conjugation	Unconjugated
Isotype	IgG
Formulation	PBS with 0.02% sodium azide, 50% glycerol, pH7.3.
Storage Instruction	Store at -20C. Avoid freeze / thaw cycles.
Database Links	HGNC:11850OMIM:603030Reactome:R-HSA-1236974
Alternative Names	Toll-like receptor 4 hToll CD antigen CD284
Function	Cooperates with LY96 and CD14 to mediate the innate immune response to bacterial lipopolysaccharide (LPS) ,
Cellular Localization	Cell membrane,
Post-translational Modifications	N-glycosylated, Glycosylation of Asn-526 and Asn-575 seems to be necessary for the expression of TLR4 on the cell surface and the LPS-response, Likewise, mutants lacking two or more of the other N-glycosylation sites were deficient in interaction with LPS,