

Immunotag™ Toll-like Receptor 3 mouse mAb

Antibody Specification	
Catalog No.	ITM1506
Product Description	Immunotag™ Toll-like Receptor 3 mouse mAb
Size	50 µg, 100 µg
Conjugation	HRP, Biotin, FITC, Alexa Fluor® 350, Alexa Fluor® 405, Alexa Fluor® 488, Alexa Fluor® 555, Alexa Fluor® 594, Alexa Fluor® 647
IMPORTANT NOTE	This product is custom manufactured with a lead time of 3-4 weeks. Once in production, this item cannot be cancelled from an order and is not eligible for return.
Target Protein	Toll-like Receptor 3
Clonality	Monoclonal
Storage/Stability	-20°C/1 year
Application	WB
Recommended Dilution	wb dilution 1:1000
Concentration	1 mg/ml
Reactive Species	Transfected
Host Species	Mouse
Immunogen	Purified recombinant human Toll-Like Receptor 3 protein fragments expressed in E.coli.
Specificity	Transfected Only.
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen
Form	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Gene Name	tlr3
Accession No.	O15455 Q99MB1
Alternate Names	CD283 ; CD283 antigen ; IIAE2 ; TLR 3 ; Tlr3 ; TLR3_HUMAN ; Toll Like Receptor 3 ; Toll-like receptor 3.

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Description	toll like receptor 3(TLR3) Homo sapiens 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 receptor is most abundantly expressed in placenta and pancreas, and is restricted to the dendritic subpopulation of the leukocytes. It recognizes dsRNA associated with viral infection, and induces the activation of NF-kappaB and the production of type I interferons. It may thus play a role in host defense against viruses. Use of alternative polyadenylation sites to generate
Cell Pathway/ Category	Toll_Like,
Protein Expression	Liver,Lung,Neuron,Placenta,
Subcellular Localization	Golgi membrane,intracellular,cytoplasm,lysosomal membrane,early endosome,endoplasmic reticulum membrane,integral component of plasma membrane,cell surface,endosome membrane,membrane,integral component of membrane,endolysosome me
Protein Function	disease:Defects in TLR3 are the cause of TLR3-deficient herpes simplex encephalitis (HSE) [MIM:603029]. HSE is a rare complication of human herpesvirus 1 (HHV-1) infection, occurring in only a small minority of HHV-1 infected individuals. HSE is characterized by hemorrhagic necrosis of parts of the temporal and frontal lobes. Onset is over several days and involves fever, headache, seizures, stupor, and often coma, frequently with a fatal outcome.,disease:Genetic variation in TLR3 is associated with susceptibility to progression to geographic atrophy in age-related macular degeneration [MIM:612479]. Age-related macular degeneration (ARMD) is the most common cause of irreversible vision loss in the developed world. In most patients, the disease is manifest as ophthalmoscopically visible yellowish accumulations of protein and lipid (known as drusen) that lie beneath the retinal pigment epithelium and within an elastin-containing structure known as Bruch's membrane. ARMD is likely to be a mechanistically heterogeneous group of disorders, and the specific disease mechanisms that underlie the vast majority of cases are currently unknown. However, a number of studies have suggested that both genetic and environmental factors are likely to play a role. Geographic atrophy (extensive atrophy of the retinal pigment epithelium and overlying photoreceptors) is an advanced form of 'dry' (nonneovascular or nonexudative) ARMD.,function:Participates in the innate immune response to microbial agents. Mediates the innate immune response to ds-RNA, a sign of viral infection. Acts via MYD88 and TRAF6, leading to NF-kappa-B activation, cytokine secretion and the inflammatory response.,online information:TLR3 mutation db,PTM:Heavily N-glycosylated, except on that part of the surface of the ectodomain that is involved in ligand binding.,similarity:Belongs to the Toll-like receptor family.,similarity:Contains 1 TIR domain.,similarity:Contains 22 LRR (leucine-rich) repeats.,subunit:Binds MYD88 via their respective TIR domains (By similarity). Interacts with TICAM1. Homodimer formation is triggered by ligand binding and is required for TLR3 signaling. Binding of ds-RNA is required for the interaction with SRC.,tissue specificity:Expressed at high level in placenta and pancreas. Also detected in CD11c+ immature dendritic cells. Only expressed in dendritic cells and not in other leukocytes, including monocyte precursors. TLR3 is the TLR that is expressed most strongly in the brain, especially in astrocytes, glia, and neurons.,

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Usage	For Research Use Only! Not for diagnostic or therapeutic procedures.
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