

TLR7 Rabbit pAb

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Catalog # AP52331

Product Information

Application	WB
Primary Accession	Q9NYK1
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Calculated MW	120922
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from human TLR7
Epitope Specificity	461-560/1049
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SUBCELLULAR LOCATION	Endoplasmic reticulum membrane; Single-pass type I membrane protein. Endosome. Lysosome. Cytoplasmic vesicle, phagosome. Note=Relocalizes from endoplasmic reticulum to endosome and lysosome upon stimulation with agonist.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	Key component of innate and adaptive immunity. TLRs (Toll-like receptors) control host immune response against pathogens through recognition of molecular patterns specific of microorganisms. TLR7 is a nucleotide-sensing TLR which is activated by single-stranded RNA. Acts via MYD88 and TRAF6, leading to NF-kappa-B activation, cytokine secretion and the inflammatory response.

Additional Information

Gene ID	51284
Other Names	Toll-like receptor 7, TLR7 (HGNC:15631)
Dilution	WB=1:500-2000
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	TLR7 (HGNC:15631)
Function	Endosomal receptor that plays a key role in innate and adaptive immunity (PubMed: 14976261 , PubMed: 32433612). Controls host immune response against pathogens through recognition of uridine- containing single strand RNAs (ssRNAs) of viral origin or guanosine analogs (PubMed: 12738885 , PubMed: 27742543 , PubMed: 31608988 , PubMed: 32706371 , PubMed: 35477763). Upon binding to agonists, undergoes dimerization that brings TIR domains from the two molecules into direct contact, leading to the recruitment of TIR-containing downstream adapter MYD88 through homotypic interaction (PubMed: 27742543). In turn, the Myddosome signaling complex is formed involving IRAK4, IRAK1, TRAF6, TRAF3 leading to activation of downstream transcription factors NF-kappa-B and IRF7 to induce pro-inflammatory cytokines and interferons, respectively (PubMed: 27742543 , PubMed: 32706371). In plasmacytoid dendritic cells, RNASET2 endonuclease cooperates with PLD3 or PLD4 5'->3' exonucleases to process RNA and release 2',3'-cyclic guanosine monophosphate (2',3'-cGMP) and cytidine-rich RNA fragments that occupy TLR7 ligand-binding pockets and trigger a signaling-competent state.
Cellular Location	Endoplasmic reticulum membrane {ECO:0000250 UniProtKB:P58681}; Single-pass type I membrane protein {ECO:0000250 UniProtKB:P58681}. Endosome {ECO:0000250 UniProtKB:P58681}. Lysosome {ECO:0000250 UniProtKB:P58681}. Cytoplasmic vesicle, phagosome {ECO:0000250 UniProtKB:P58681}. Note=Relocalizes from endoplasmic reticulum to endosome and lysosome upon stimulation with agonist {ECO:0000250 UniProtKB:P58681}
Tissue Location	Detected in brain, placenta, spleen, stomach, small intestine, lung and in plasmacytoid pre-dendritic cells. Expressed in peripheral mononuclear blood cells (PubMed:32706371)

Background

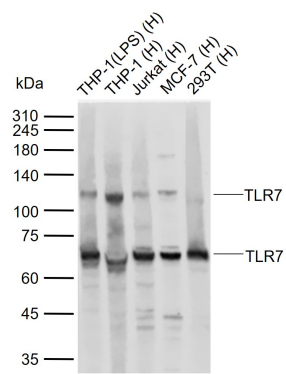
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References

Du X.,et al.Eur. Cytokine Netw. 11:362-371(2000).
Chuang T.-H.,et al.Eur. Cytokine Netw. 11:372-378(2000).
Georgel P.,et al.PLoS ONE 4:E7803-E7803(2009).
Clark H.F.,et al.Genome Res. 13:2265-2270(2003).
Gantier M.P.,et al.J. Immunol. 180:2117-2124(2008).

Images

Sample:
Lane 1: Human THP-1(LPS) cell lysates
Lane 2: Human THP-1 cell lysates
Lane 3: Human Jurkat cell lysates
Lane 4: Human MCF-7 cell lysates
Lane 5: Human 293T cell lysates
Primary: Anti-TLR7 (AP52331) at 1/1000 dilution
Secondary: IRDye800CW Goat Anti-Rabbit IgG at 1/20000



dilution

Predicted band size: 121 kDa

Observed band size: 65,121 kDa

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.