

## Rabbit Anti-phospho-IRAK4 (Thr345) antibody

SL10208R

phospho-IRAK4 (Thr345)
磷酸化白介素-1受体相关激酶4抗体
IL-1 Receptor-associated Kinase 4; 8430405M07Rik; 9330209D03Rik; IPD1; IRAK4; NY-REN-64; REN64; INTERLEUKIN RECEPTOR-ASSOCIATED KINASE 4; Interleukin 1 receptor associated kinase 4 mutant form 1; Interleukin-1 receptor- associated kinase 4; Interleukin1 receptor associated kinase 4; IPD1; IRAK 4; IRAK-4; IRAK4 mutated form 1; IRAK4_HUMAN; LOC 51135; NY REN 64 antigen; Renal carcinoma antigen NY-REN-64.
Rabbit
Polyclonal
Human, Mouse, Rat, Dog, Pig, Cow, Rabbit, Sheep, Guinea Pig,
WB=1:500-2000ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800IF=1:100- 500 (Paraffin sections need antigen repair) not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
51kDa
cytoplasmic
Lyophilized or Liquid
lmg/ml
KLH conjugated Synthesised phosphopeptide derived from human IRAK4 around the phosphorylation site of Thr345:VM(p-T)SR
IgG
affinity purified by Protein A
0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20°C. 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.
PubMed

	This gene encodes a kinase that activates NF-kappaB in both the Toll-like receptor (TLR) and T-cell receptor (TCR) signaling pathways. The protein is essential for most innate immune responses. Mutations in this gene result in IRAK4 deficiency and recurrent invasive pneumococcal disease. Multiple transcript variants encoding different
	isoforms have been found for this gene. [provided by RefSeq, Aug 2011]
Product Detail:	<ul> <li>Function:</li> <li>Serine/threeonine-protein kinase that plays a critical role in initiating innate immune response against foreign pathogens. Involved in Toll-like receptor (TLR) and IL-1R signaling pathways. Is rapidly recruited by MYD88 to the receptor-signaling complex upon TLR activation to form the Myddosome together with IRAK2. Phosphorylates initially IRAK1, thus stimulating the kinase activity and intensive autophosphorylation of IRAK1. Phosphorylates E3 ubiquitin ligases Pellino proteins (PEL11, PEL12 and PEL13) to promote pellino-mediated polyubiquitination of IRAK1. Then, the ubiquitin-binding domain of IKBKG/NEMO binds to polyubiquitinated IRAK1 bringing together the IRAK1-MAP3K7/TAK1-TRAF6 complex and the NEMO-IKKA-IKKB complex. In turn, MAP3K7/TAK1 activates IKKs (CHUK/IKKA and IKBKB/IKKB) leading to NF-kappa-B nuclear translocation and activation. Alternatively, phosphorylates TIRAP to promote its ubiquitination and subsequent degradation. Phosphorylates NCF1 and regulates NADPH oxidase activation after LPS stimulation suggesting a similar mechanism during microbial infections.</li> <li>Subunit:</li> <li>Associates with MYD88 and IRAK2 to form a ternary complex called the Myddosome. Once phosphorylated, IRAK4 dissociates from the receptor complex and then associates with the TNF receptor-associated factor 6 (TRAF6), IRAK1, and PEL11; this intermediate complex is required for subsequent NF-kappa-B activation. Interacts with ILIRL1.</li> <li>Subcellular Location: Cytoplasm.</li> </ul>
	Post-translational modifications:
	Phosphorylated.
	<b>DISEASE:</b> Defects in IRAK4 are the cause of recurrent isolated invasive pneumococcal disease type 1 (IPD1) [MIM:610799]. Recurrent invasive pneumococcal disease (IPD) is defined as two episodes of IPD occurring at least 1 month apart, whether caused by the same or different serotypes or strains. Recurrent IPD occurs in at least 2% of patients in most series, making IPD the most important known risk factor for subsequent IPD. Defects in IRAK4 are the cause of IRAK4 deficiency (IRAK4D) [MIM:607676]. IRAK4 deficiency causes extracellular pyogenic bacterial and fungal infections in otherwise healthy children.
	Similarity:





