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IRAK Antibody (N-term) Blocking peptide
Synthetic peptide
Catalog # BP7802a

Specification

IRAK Antibody (N-term) Blocking peptide - Product Information

Primary Accession P51617

IRAK Antibody (N-term) Blocking peptide - Additional Information

Gene ID 3654

Other Names
Interleukin-1 receptor-associated kinase 1, IRAK-1, IRAK1, IRAK

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP7802a was selected from the N-term region of human IRAK1 . 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.

IRAK Antibody (N-term) Blocking peptide - Protein Information

Name IRAK1 (HGNC:6112)

Synonyms IRAK

Function
Serine/threonine-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. Association with MYD88 leads to IRAK1 phosphorylation by IRAK4 and subsequent autophosphorylation and kinase activation. Phosphorylates E3 ubiquitin ligases Pellino proteins (PELI1, PELI2 and PELI3) 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

the interferon regulatory factor 7 (IRF7) to induce its activation and translocation to the nucleus, resulting in transcriptional activation of type I IFN genes, which drive the cell in an antiviral state. When sumoylated, translocates to the nucleus and phosphorylates STAT3.

Cellular Location
Cytoplasm. Nucleus. Lipid droplet Note=Translocates to the nucleus when sumoylated. RSAD2/viperin recruits it to the lipid droplet (By similarity).

Tissue Location
Isoform 1 and isoform 2 are ubiquitously expressed in all tissues examined, with isoform 1 being more strongly expressed than isoform 2.

IRAK Antibody (N-term) Blocking peptide - Protocols

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

- Blocking Peptides

IRAK Antibody (N-term) Blocking peptide - Images

IRAK Antibody (N-term) Blocking peptide - Background

Protein kinases are enzymes that transfer a phosphate group from a phosphate donor, generally the g phosphate of ATP, onto an acceptor amino acid in a substrate protein. By this basic mechanism, protein kinases mediate most of the signal transduction in eukaryotic cells, regulating cellular metabolism, transcription, cell cycle progression, cytoskeletal rearrangement and cell movement, apoptosis, and differentiation. With more than 500 gene products, the protein kinase family is one of the largest families of proteins in eukaryotes. The family has been classified in 8 major groups based on sequence comparison of their tyrosine (PTK) or serine/threonine (STK) kinase catalytic domains. The tyrosine-like kinase (TLK) group consists of 40 tyrosine and serine-threonine kinases such as MLK (mixed-lineage kinase), LISK (LIMK/TESK), IRAK (interleukin-1 receptor-associated kinase), Raf, RIPK (receptor-interacting protein kinase), and STRK (activin and TGF-beta receptors) families.

IRAK Antibody (N-term) Blocking peptide - References

Jensen, L.E., et al., J. Immunol. 171(3):1500-1506 (2003).Jiang, Z., et al., J. Biol. Chem. 278(13):10952-10956 (2003).Cao, Z., et al., Science 271(5252):1128-1131 (1996).Strelow, A., et al., FEBS Lett. 547 (1-3), 157-161 (2003).