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TOLLIP

Reactivity: Human Mouse Rat

Tested applications:WB IHC IF

Recommended Dilution:WB 1:500 - 1:2000 IHC 1:50 - 1:200 IF 1:50 - 1:200

Calculated MW:30kDa

Observed MW: Refer to Figures

Immunogen:

Recombinant protein of human TOLLIP

Storage Buffer:

Store at -20. Avoid freeze / thaw cycles. Buffer: PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

Concentration:

s

Synonym:

FLJ33531; IL-1RAcPIP;

Catalog #:A2202

Antibody Type:

Polyclonal Antibody

Species: Rabbit

Gene ID:54472

Isotype: IgG

Swiss Prot:Q9H0E2

Purity: Affinity purification

For research use only.

Background:

innate immune responses (1-3). TLRs recognize conserved motifs found in various pathogens and mediate defense responses. Triggering of the TLR pathway leads to the activation of NF-B and subsequent regulation of immune and inflammatory genes. The TLRs and members of the IL-1 receptor family share a conserved stretch of approximately 200 amino acids known as the Toll/Interleukin-1 receptor (TIR) domain. Upon activation, TLRs associate with a number of cytoplasmic adaptor proteins containing TIR domains, including myeloid differentiation factor 88 (MyD88), MyD88-adaptor-like/TIR-associated protein (MAL/TIRAP), Toll-receptor-associated activator of interferon (TRIF), and Toll-receptor-associated molecule (TRAM). This association leads to the recruitment and activation of IRAK1 and IRAK4, which form a complex with TRAF6 to activate TAK1 and IKK. Activation of IKK leads to the degradation of IB, which normally maintains NF-B in an inactive state by sequestering it in the cytoplasm.Tollip (Toll interacting protein) is an adaptor protein discovered to be associated with the IRAK complex and recruited to IL1-R following IL-1 stimulation (4). Overexpression of Tollip results in impaired NF-B signaling (4). Tollip also associates directly with TLR2 and TLR4 and inhibits TLR-mediated signaling through inhibition of IRAK (5). Studies of Tollip deficient mice suggest that it plays a role in the regulation of inflammatory cytokines in response to IL-1 and LPS (6).

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