

MyD88

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:33kDa

Observed MW:Refer to Figures

Immunogen:

Recombinant protein of human MyD88

Storage Buffer:

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

Concentration:

ps

Synonym:

MYD88D

Catalog #:A0980

Antibody Type:

Polyclonal Antibody

Species:Rabbit

Gene ID:4615

Isotype:IgG

Swiss Prot:Q99836

Purity:Affinity purification

For research use only.

Background:

Members of the Toll-like receptor (TLR) family, named for the closely related Toll receptor in *Drosophila*, play a pivotal role in 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- κ 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 TIR domain. Upon activation, TLRs associate with a number of cytoplasmic adaptor proteins containing TIR domains including MyD88 (myeloid differentiation factor), MAL/TIRAP (MyD88-adaptor-like/TIR-associated protein), TRIF (Toll-receptor-associated activator of interferon), and TRAM (Toll-receptor-associated molecule). 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 I κ B that normally maintains NF- κ B inactivity by sequestering it in the cytoplasm. MyD88 was originally isolated as a myeloid differentiation primary response gene that is rapidly induced upon IL-6 stimulated differentiation of M1 myeloleukemic cells into macrophages (4-6). It contains an amino-terminal death domain separated from a carboxyl-terminal TIR domain and functions as an adaptor in TLR/IL-1 receptor signaling (7). The death domain of MyD88 mediates interactions with the IRAK complex triggering a signaling cascade that includes the activation of NF- κ B (8,9).

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