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# 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-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 IB that normally maintains NF-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-B (8,9).

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