MyD88 Antibody

For Research Use Only. Not For Use In Diagnostic Procedures.

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 (1-4). TLRs recognize conserved motifs found in various pathogens and mediate defense responses (5-7). Triggering of the TLR pathway leads to the activation of NF-κB and subsequent regulation of immune and inflammatory genes (4). 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 (1). 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) (8-10). This association leads to the recruitment and activation of IRAK1 and IRAK4, which form a complex with TRAF6 to activate TAK1 and IKK (8,11-14). Activation of IKK leads to the degradation of IκB, which normally maintains NF-κB in an inactive state 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 (15-17). It contains an amino-terminal death domain separated from a carboxyl-terminal TIR domain 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 (1). 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) (8-10). This association leads to the recruitment and activation of IRAK1 and IRAK4, which form a complex with TRAF6 to activate TAK1 and IKK (8,11-14). Activation of IKK leads to the degradation of IκB, which normally maintains NF-κB in an inactive state 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 (15-17). 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 (18). The death domain of MyD88 mediates interactions with the IRAK complex triggering a signaling cascade that includes the activation of NF-κB (19, 20).

Specificity/Sensitivity: MyD88 (D80F5) Rabbit mAb detects endogenous levels of total MyD88 protein.

Source/Purification: Monoclonal antibody is prepared from animals immunized with a synthetic peptide corresponding to residues surrounding Cys233 of human MyD88 protein.

Recommended Antibody Dilutions:
Western blotting 1:1000

IMPORTANT: For western blots, incubate membrane with diluted antibody in 5% w/v BSA, 1X TBS, 0.1% Tween-20 at 4°C with gentle shaking, overnight.

Applications

- Western
- Immunoprecipitation
- ELISA-Peptide

Species Cross-Reactivity Key:

- H—human
- M—mouse
- R—rat
- Hm—hamster
- Mk—monkey
- Mi—mink
- C—chicken
- Dm—D. melanogaster
- X—Xenopus
- Z—zebrafish
- B—bovine
- S. cerevisiae
- C. elegans
- H—horse
- A—all species expected

Storage: Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at ~20°C. Do not aliquot the antibody.

*Species cross-reactivity is determined by western blot.

**Anti-rabbit secondary antibodies must be used to detect this antibody.

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Background References: