Revision 1

Store at -20C

#50010

MyD88 (E9K4E) Mouse mAb

Cell Signaling TECHNOLOGY®

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	H	Endogenous	33	Mouse IgG1	#Q99836	4615

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:50

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

MyD88 (E9K4E) Mouse mAb recognizes endogenous levels of total MyD88 protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Leu50 of human MyD88 protein.

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 adapter proteins containing TIR domains, including myeloid differentiation factor 88 (MyD88), MyD88-adapter-like/TIR-associated protein (MAL/TIRAP), TIR domain-containing adapter-inducing IFN-β (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).

Background References

1. Akira, S. (2003) *J Biol Chem* 278, 38105-8.
2. Beutler, B. (2004) *Nature* 430, 257-63.
3. Dunne, A. and O'Neill, L.A. (2003) *Sci STKE* 2003, re3.
4. Medzhitov, R. et al. (1997) *Nature* 388, 394-7.
5. Schwandner, R. et al. (1999) *J Biol Chem* 274, 17406-9.
6. Takeuchi, O. et al. (1999) *Immunity* 11, 443-51.
7. Alexopoulou, L. et al. (2001) *Nature* 413, 732-8.
8. Zhang, F.X. et al. (1999) *J Biol Chem* 274, 7611-4.
9. Horng, T. et al. (2001) *Nat Immunol* 2, 835-41.
10. Oshiumi, H. et al. (2003) *Nat Immunol* 4, 161-7.
11. Muzio, M. et al. (1997) *Science* 278, 1612-5.
12. Wesche, H. et al. (1997) *Immunity* 7, 837-47.
13. Suzuki, N. et al. (2002) *Nature* 416, 750-6.
14. Irie, T. et al. (2000) *FEBS Lett* 467, 160-4.
15. Harroch, S. et al. (1995) *Nucleic Acids Res* 23, 3539-46.
16. Hardiman, G. et al. (1996) *Oncogene* 13, 2467-75.
17. Bonnert, T.P. et al. (1997) *FEBS Lett* 402, 81-4.
18. Medzhitov, R. et al. (1998) *Mol Cell* 2, 253-8.
19. Wesche, H. et al. (1997) *Immunity* 7, 837-47.
20. Muzio, M. et al. (1997) *Science* 278, 1612-5.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

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

Applications Key

W: Western Blotting **IP:** Immunoprecipitation

Cross-Reactivity Key

H: Human

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Limited Uses