

TICAM1

Reactivity: Human Mouse Rat

Tested applications: WB IHC

Recommended Dilution: WB 1:500 - 1:2000 IHC 1:50 - 1:100

Calculated MW: 76kDa

Observed MW: Refer to Figures

Immunogen:

Recombinant protein of human TICAM1

Storage Buffer:

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

Concentration:

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Synonym:

TRIF; PRVTIRB; TICAM-1

Catalog #: A1155

Antibody Type:

Polyclonal Antibody

Species: Rabbit

Gene ID: 148022

Isotype: IgG

Swiss Prot: Q8IUC6

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. TRIF (also termed TICAM-1) is a TIR-domain adaptor protein described to activate NF- κ B, IRF3 and trigger IFN- γ production (4,5). Studies using dominant negative forms of TRIF and siRNA targeting TRIF show that TRIF functions downstream of both TLR3 and TLR4 in response to dsRNA and LPS respectively (4-6). TRIF recruits TRAF6-TAK1-TAB2 to the receptor complex which leads to NF- κ B activation (7). In addition, TRIF can trigger signaling of that lead to the induction of apoptosis (8).

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