

## TLR3

**Reactivity:** Human Mouse Rat

**Tested applications:** WB IHC

**Recommended Dilution:** WB 1:500 - 1:2000 IHC 1:50 - 1:200

**Calculated MW:** 99kDa

**Observed MW:** Refer to Figures

**Immunogen:**

Recombinant protein of human TLR3

**Storage Buffer:**

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

**Concentration:**

5

**Synonym:**

TLR3;CD283 ;

**Catalog #:** A1449

**Antibody Type:**

Polyclonal Antibody

**Species:** Rabbit

**Gene ID:** 7098

**Isotype:** IgG

**Swiss Prot:** O15455

**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- $\kappa$ 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 $\kappa$ B that normally maintains NF- $\kappa$ B inactivity by sequestering it in the cytoplasm. TLR3 functions as a receptor for double-stranded (ds)RNA typically associated with viral infection (4). It was originally shown to be specifically expressed in dendritic cells of the leukocyte family (5). TLR3 has also been found in placenta and lung, and can be induced by LPS in a variety of tissues (4,6). TLR3 is predominantly localized to early endosomes (7,8). Binding of dsRNA, or the analog polyinosine-polycytidylic acid (plpC), to TLR3 triggers activation of transcription factors NF- $\kappa$ B and IRF3 through the adaptor protein TICAM-1/TRIF (9,10). TRIF associates with members of the TRAF family and with RIP that combine to activate NF- $\kappa$ B and IRF3 (11-13).

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