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| Product name: | TNF-R1 Rabbit Polyclonal Antibody |
| Cat number: | ABN19092 |
| Conjugate: | Unconjugated |
| Size: | 100µL |
| Clone: | Polyclonal |
| Concentration: | 1mg/ml |
| Host: | Rabbit |
| Isotype: | IgG |
| Immunogen: | The antiserum was produced against synthesized peptide derived from human TNF Receptor I. AA range:381-430 |
| Reactivity: | Human,Mouse,Rat |
| Applications: | WB 1:500-1:2000,IHC 1:100-1:300,ICC/IF 1:50-1:200,ELISA 1:10000-1:20000 |
| Molecular Weight: | 50kDa |
| Purification: | Affinity purification |
| Form: | Liquid |
| Buffer: | Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N. |
| Storage: | Store at 4°C short term. Aliquot and store at -20°C for 12 months. Avoid freeze/thaw cycles. |

Background:

This gene encodes a member of the TNF receptor superfamily of proteins. The encoded receptor is found in membrane-bound and soluble forms that interact with membrane-bound and soluble forms, respectively, of its ligand, tumor necrosis factor alpha. Binding of membrane-bound tumor necrosis factor alpha to the membrane-bound receptor induces receptor trimerization and activation, which plays a role in cell survival, apoptosis, and inflammation. Proteolytic processing of the encoded receptor results in release of the soluble form of the receptor, which can interact with free tumor necrosis factor alpha to inhibit inflammation. Mutations in this gene underlie tumor necrosis factor receptor-associated periodic syndrome (TRAPS), characterized by fever, abdominal pain and other features. Mutations in this gene may also be associated with multiple sclerosis in human patients. [provided by Redisease:Defects in TNFRSF1A are the cause of familial hibernian fever (FHF) [MIM:142680]; also known as tumor necrosis factor receptor-associated periodic syndrome (TRAPS). FHF is a hereditary periodic fever syndrome characterized by recurrent fever, abdominal pain, localized tender skin lesions and myalgia. Reactive amyloidosis is the main complication and occurs in 25% of cases.,domain:Both the cytoplasmic membrane-proximal region and the C-terminal region containing the death domain are involved in the interaction with TRPC4AP.,domain:The domain that induces A-SMASE is probably identical to the death domain. The N-SMASE activation domain (NSD) is both necessary and sufficient for activation of N-SMASE.,function:Receptor for TNFSF2/TNF-alpha and homotrimeric TNFSF1/lymphotoxin-alpha. The adapter molecule FADD recruits caspase-8 to the activated receptor. The resulting death-inducing signaling complex (DISC) performs caspase-8 proteolytic activation which initiates the subsequent cascade of caspases (aspartate-specific cysteine proteases) mediating apoptosis. Contributes to the induction of non-cytocidal TNF effects including anti-viral state and activation of the acid sphingomyelinase.,online information:Repertory of FMF and hereditary autoinflammatory disorders mutations,PTM:The soluble form is produced from the membrane form by proteolytic processing.,similarity:Contains 1 death domain.,similarity:Contains 4 TNFR-Cys repeats.,subunit:Binding of TNF to the extracellular domain leads to homotrimerization. The aggregated death domains provide a novel molecular interface that interacts specifically with the death domain of TRADD. Various TRADD-interacting proteins such as TRAFs, RIPK1 and possibly FADD, are recruited to the complex by their association with TRADD. This complex activates at least two distinct signaling cascades, apoptosis and NF-kappa-B signaling. Interacts with BAG4, BRE, FEM1B, GRB2, SQSTM1 and TRPC4AP. Interacts with HCV core protein.,