

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse TNF RII/TNFRSF1B in direct ELISAs.
Source	Monoclonal Rat IgG ₁ Clone # 91026R
Purification	Protein A or G purified from cell culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant mouse TNF RII/TNFRSF1B Val23-Gly258 Accession # Q545P4
Conjugate	Alexa Fluor 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

ELISA Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Tumor Necrosis Factor Receptor II (TNF RII), also known as TNFRSF1B, p75/p80, and CD120b, is a type I transmembrane protein that belongs to the TNF receptor superfamily. It has a molecular weight of approximately 75 kDa (1-4). The TNF receptor superfamily is comprised of structurally related receptors that bind to TNF-related ligands and regulate numerous processes such as immune cell activation and apoptosis. Receptors in this superfamily are characterized by the presence of a cysteine-rich region in their extracellular domain (ECD) (1-3, 5). Mouse TNF RII contains four cysteine-rich repeats in its ECD, which shares 58% and 84% amino acid sequence identity with the human and rat orthologs, respectively. Several receptors in the TNF superfamily also contain intracellular death domains (DDs) that recruit caspase-interacting proteins to initiate apoptosis upon ligand binding. Those receptors that lack DDs, like TNF RII, bind TNF Receptor-associated Factors, which transduce signals generated by activation of these receptors (6, 7).

TNF RII is expressed predominantly on cells of the hematopoietic lineage, such as T and natural killer cells, as well as on endothelial cells, microglia, astrocytes, neurons, oligodendrocytes, cardiac myocytes, and thymocytes (6, 8, 9). In humans, TNF RII is also located on mesenchymal stem cells (6, 9, 10). TNF RII binds to the membrane-bound forms of TNF-α and Lymphotoxin-α/TNF-β; soluble TNF is thought to signal predominately through TNF RI (7, 11). TNF RII activation primarily initiates pro-inflammatory and pro-survival responses via NFκB-dependent signaling pathways (6, 7, 12-15). However, under certain conditions, TNF RII signaling can induce apoptosis (6). TNF RII also exists as a soluble receptor, which can be generated by proteolytic cleavage of its ECD from the cell surface or by alternative splicing (2, 16). Soluble TNF RII is believed to inhibit TNF biological activity by binding TNF thereby preventing it from activating membrane TNF receptors (17). Polymorphisms of the human *TNFR2* gene, which result in increased expression of both membrane-bound and soluble TNF RII, have been associated with several autoimmune diseases including Crohn's disease, systemic lupus erythematosus, and familial rheumatoid arthritis (6, 17).

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