Recombinant Mouse TNF RII/TNFRSF1B

Catalog Number: 426-R2/CF

DESCRIPTION

Source	*E. coli*-derived Val23-Gly258 Accession # Q545P4
N-terminal Sequence Analysis	Val23
Predicted Molecular Mass	25 kDa

SPECIFICATIONS

Activity	Measured by its ability to inhibit the TNF-α mediated cytotoxicity in the L-929 mouse fibroblast cells in the presence of the metabolic inhibitor actinomycin D. Matthews, N. and M.L. Neale (1987) in Lymphokines and Interferons, A Practical Approach. Clemens, M.J. *et al.* (eds): IRL Press. 221. The ED_{50} for this effect is 1-3 µg/mL in the presence of 0.1 ng/mL of recombinant mouse TNF-α.
Endotoxin Level	<0.01 EU per 1 µg of the protein by the LAL method.
Purity	>97%, by SDS-PAGE under reducing conditions and visualized by silver stain.
Formulation	Lyophilized from a 0.2 µm filtered solution in Acetonitrile and TFA. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 100 µg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

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).

References:

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