

Recombinant Rat Fas/TNFRSF6/CD95 Protein(Fc Tag)

Catalog Number: PDMR100072

Note: *Centrifuge before opening to ensure complete recovery of vial contents.*

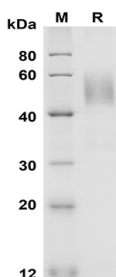
Description

Species	Rat
Source	Mammalian-derived Rat Fas/TNFRSF6/CD95 proteins Gln22-Leu171, with an C-terminal Fc
Calculated MW	41.4 kDa
Observed MW	50 kDa
Accession	Q63199
Bio-activity	Not validated for activity

Properties

Purity	> 90% as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU/mg of the protein as determined by the LAL method
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

Data



SDS-PAGE analysis of Rat Fas/TNFRSF6/CD95 proteins, 2 µg/lane of Recombinant Rat Fas/TNFRSF6/CD95 proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 50 kDa.

Background

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CD95 (APO-1/Fas) is an important inducer of the extrinsic apoptosis signaling pathway and therapy induced apoptosis of many tumor cells has been linked to the activity of CD95. is a prototype death receptor characterized by the presence of an 80 amino acid death domain in its cytoplasmic tail. This domain is essential for the recruitment of a number of signaling components upon activation by either agonistic anti-CD95 antibodies or cognate CD95 ligand that initiate apoptosis. The complex of proteins that forms upon triggering of CD95 is called the death-inducing signaling complex (DISC). The DISC consists of an adaptor protein and initiator caspases and is essential for induction of apoptosis. CD95 is also crucial for the negative selection of B cells within the germinal center (GC). Impairment of CD95-mediated apoptosis results in defective affinity maturation and the persistence of autoreactive B-cell clones. Changes in the expression of CD95 and/or its ligand CD95L are frequently found in human cancer. The downregulation or mutation of CD95 has been proposed as a mechanism by which cancer cells avoid destruction by the immune system through reduced apoptosis sensitivity. Thus, CD95 has therefore been viewed as a tumor suppressor. CD95 has been reported to be involved in the activation of NF-kappaB, MAPK3/ERK1, MAPK8/JNK, and the alternate pathways for CTL-mediated cytotoxicity. Accordingly, this protein is implicated in the pathogenesis of various malignancies and diseases of the immune system. The CD95/CD95L system was implicated in the etiology of inflammatory bowel disease (IBD) based, primarily, on the finding that CD95 is highly expressed in the intestinal epithelial cells and that epithelial apoptosis is increased in IBD.

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