

Recombinant Mouse FAS/TNFRSF6 Protein (His Tag)

Catalog Number: PKSM040905

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

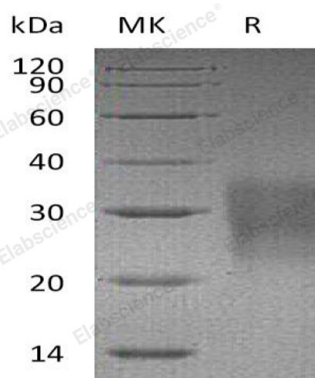
Description

Species	Mouse
Source	HEK293 Cells-derived Mouse FAS/TNFRSF6 protein Met 1-Arg 169, with an C-terminal His
Calculated MW	18.0 kDa
Accession	NP_032013.2
Bio-activity	Measured by its ability to inhibit Fas Ligand induced apoptosis of Jurkat human acute T cell leukemia cells. The ED ₅₀ for this effect is typically 1-8 µg/mL in the presence of 20 ng/mL recombinant human Fas ligand.

Properties

Purity	> 98 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per µg 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 sterile PBS, pH 7.4 Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



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Background

For Research Use Only

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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. 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.