Recombinant Mouse EDAR/DL Protein (Fc Tag)

Catalog Number: PKSM041006

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

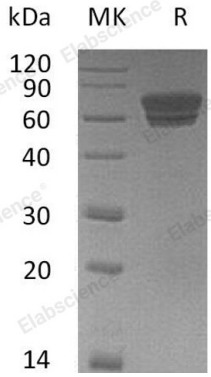
Description

Species	Mouse
Mol_Mass	44.7 kDa
Accession	Q9R187
Bio-activity	Not validated for activity

Properties

Purity	> 95 % 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 ℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ 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 of 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



> 95 % as determined by reducing SDS-PAGE.

Background

Ectodysplasin A receptor (EDAR) is a type I transmembrane protein of the TNF-α receptor superfamily which plays a key role in ectodermal differentiation. EDAR was encoded by the mouse downless gene and defective in human dominant and recessive forms of autosomal hypohidrotic ectodermal dysplasia (EDA) syndrome. The extracellular domain of EDAR contains 14 cysteine residues, six of which approximate the TNFRSF cysteine-rich region, the cytoplasmic domain contains a region with homology to the death domains found in other TNFRSF members. EDAR has been suggested to be an early and important promoter of placode development in all ectodermal organs, such as uch as hair follicles, teeth and sweat glands. EDA-A1, the A1 isoform of EDA, is the EDAR ligand. EDA and EDA are implicated in appendage development by the cloning of a gene underlying hypohidrotic ectodermal dysplasia (HED) in mouse and human. HED is characterized by agenesis or malformation of ectoderm-derived appendages, such as teeth, sweat glands and hair follicles, while the skin itself develops normally.

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