

Recombinant Mouse ALK-1/ACVRL1 Protein (Fc Tag)

Catalog No. PKSM041235

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

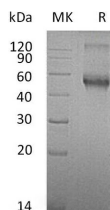
Description

Synonyms	Serine/threonine-protein kinase receptor R3;SKR3;Activin receptor-like kinase 1;ALK-1;TGF-B superfamily receptor type I;TSR-I;ACVRL1;activin A receptor type II-like 1;activin A receptor
Species	Mouse
Expression Host	HEK293 Cells
Sequence	Asp23-Pro119
Accession	Q61288
Calculated Molecular Weight	38.1 kDa
Observed molecular weight	55-60 kDa
Tag	C-Fc
Bioactivity	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°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 of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 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

For Research Use Only

Activin Receptor-Like Kinase 1 (ALK-1) is a type I cell-surface receptor for the TGF- β superfamily of ligands, which mediates signaling of BMP9 (bone morphogenetic protein) and BMP10. ALK1 signaling is necessary for angiogenesis during embryogenesis, wound healing, and tumor growth. ALK-1 has a high degree of similarity in serine-threonine kinase subdomains, a glycine and serine rich region preceding the kinase-domain, and a C-terminal tail with other activin receptor-like kinase proteins. ALK-1 is mainly expressed in endothelial cells regulating proliferation and migration in vitro and angiogenesis in vivo. Mutations in ALK-1 as well as in endoglin are associated with hereditary hemorrhagic telangiectasia (HHT), suggesting ALK-1 plays a critical role for in the control of blood vessel development or repair.