Recombinant Rat ALK-2/ACVR1 Protein (Fc Tag)

Catalog Number: PKSR030357

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

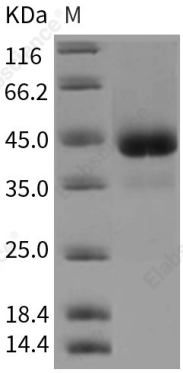
Description

Species	Rat
Source	HEK293 Cells-derived Rat ALK-2/ACVR1 protein Met1-Glu123, with an C-terminal hFc
Calculated MW	38.5 kDa
Observed MW	43 kDa
Accession	P80201
Bio-activity	Not validated for activity

Properties

Purity	> 90% 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



> 90 % as determined by reducing SDS-PAGE.

Background

ALK-2, also termed as ACVR1, was initially identified as an activin type I receptor because of its ability to bind activin in concert with ActRII or ActRIIB. ALK-2 is also identified as a BMP type I receptor. It has been demonstrated that ALK-2 forms complex with either the BMP-2/7-bound BMPR-II or ACVR2A/ACVR2B. ALK-1 and ALK-2 presenting in the yeast Saccharomyces cerevisiae are two haspin homologues. Both ALK-1 and ALK-2 exhibit a weak auto-kinase activity in vitro, and are phosphoproteins in vivo. ALK-1 and ALK-2 levels peak in mitosis and late-S/G2. Control of protein stability plays a major role in ALK-2 regulation. The half-life of ALK-2 is particularly short in G1. Overexpression of ALK-2, but not of ALK-1, causes a mitotic arrest, which is correlated to the kinase activity of the protein. This suggests a role for ALK-2 in the control of mitosis. Endoglin is phosphorylated on cytosolic domain threonine residues by the TG F-beta type I receptors ALK-2 and ALK-5 in prostate cancer cells. Endoglin did not inhibit cell migration in the presence of constitutively active ALK-2. Defects in ALK-2 are a cause of fibrodysplasia ossificans progressiva (FOP).

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