Recombinant Human ALK-2/ACVR1 Protein (Baculovirus, His Tag)

Catalog Number:PKSH030419

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

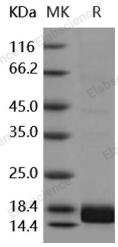
Description

Synonyms	Activin Receptor Type-1;Activin Receptor Type I;ACTR-I;Activin Receptor-Like Kinase 2;ALK-2;Serine/Threonine-Protein Kinase Receptor R1;SKR1;TGF-B Superfamily Receptor Type I;TSR-I;ACVR1;ACVRLK2;ACVR1A;ACVRLK2;ALK2;FOP;SKR1
Species	Human
Expression Host	Baculovirus-Insect Cells
Sequence	Met 1-Val 124
Accession	Q04771
Calculated Molecular Weight	12.8 kDa
Observed molecular weight	17 kDa
Tag	C-His

Properties

Purity	> 93 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < - 20°C.
Formulation	Supplied as sterile solution of 20mM Tris, 500mM NaCl, pH 7.4, 10% glycerol
Reconstitution	Not Applicable

Data



> 93 % 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 TGF-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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