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Elabscience Biotechnology Inc.

Elabscience®

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

Catalog No. PKSH030419

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	Met1-Val124
Accession	Q04771
Mol_Mass	12.8 kDa
AP_Mol_Mass	17 kDa
Tag	C-His

Properties

Purity	> 93 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg 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	Lyophilized from sterile 20mM Tris, 500mM NaCl, pH 7.5, 25% glycreol, 5% Trehalose, 5% Mannitol, 0.01 % Tween-80
Reconstitution	Not Applicable

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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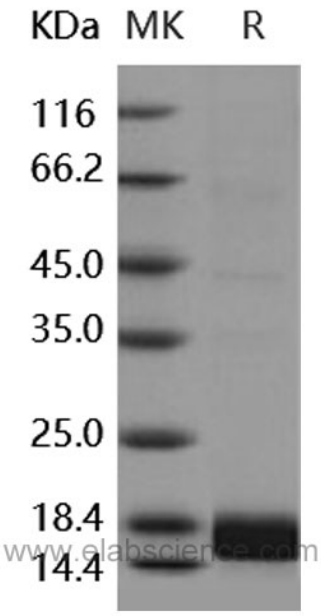
SDS-PAGE



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