Recombinant Protein Technical Manual

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

RPES1990

Product Data:

Product SKU: RPES1990

Size: 100µg

Species: Human

Expression host: Baculovirus-Insect Cells

Uniprot: Q04771

Protein Information:

Molecular Mass:	12.8 kDa
AP Molecular Mass:	17 kDa
Tag:	C-His
Bio-activity:	
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:	Supplied as sterile 20mM Tris, 500mM NaCl, pH 7.4, 10% gly
Reconstitution:	Please refer to the printed manual for detailed information.
Application:	
Synonyms:	Activin Receptor Type; 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

Immunogen Information:

Sequence: Met 1-Val 124

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 and ALK-2 presenting in the yeast Saccharomyces cerevisiae are two haspin homologues. Both ALK and ALK-2 exhibit a weak auto-kinase activity in vitro, and are phosphoproteins in vivo. ALK 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, 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).