Recombinant Protein Technical Manual

Recombinant Mouse ALK-2/ACVR1 Protein (His & Fc Tag)(Active)

RPES3040

Product Data:

Product SKU: RPES3040

Size: 100µg

Species: Mouse

Expression host: HEK293 Cells

Uniprot: NP_031420.2

Protein Information:

Molecular Mass:	40 kDa
AP Molecular Mass:	45 kDa
Tag:	C-His-Fc
Bio-activity:	Measure by its ability to bind with human BMP2 in a functional ELISA.
Purity:	> 90 % as determined by SDS-PAGE
Endotoxin:	< 1.0 EU per µg of the protein as determined by the LAL method.
Storage:	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℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ 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
Reconstitution:	Please refer to the printed manual for detailed information.
Application:	Functional ELISA
Synonyms:	ActR-I;ActRIA;Acvr;Acvrlk2;Alk-2;ALK2;Alk8;D330013D15Rik;SKR1;Tsk7L

Immunogen Information:

Sequence: Met 1-Glu 123

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).