Data Sheet (Cat.No.TMPY-04774)

TargetMol

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

General Information

Synonyms: TSRI;ACVRLK2;FOP;activin A receptor, type I;ACTRI;ACVR1A;SKR1;ALK2

Protein Construction: A DNA sequence encoding the extracellular domain (Met 1-Val 124) of human ALK2 (Q04771) (Met 1-Val 124) was fused with a polyhistidine tag at the C-terminus. Predicted N terminal: Met 21

Species: Human

Expression Host: Baculovirus Insect Cells

Accession: Q04771

Molecular Weight: 12.8kDa (predicted); 17 kDa (reducing conditions)

QC Testing

Biological Activity: Kinase activity untested

Purity: > 93 % as determined by SDS-PAGE

Endotoxin: < 1.0 EU/μg of the protein as determined by the LAL method.

Formulation: Supplied as sterile 20 mM Tris, 500 mM NaCl, pH 7.4, 10% gly.

Preparation and Storage

Reconstitution:

Reconstituted with sterile deionized water to 0.25 mg/mL. Reconstitution conditions may vary depending on the lot.

Stability & Storage:

It is recommended to store the product under sterile conditions at -20°C to -80°C. Samples are stable for up to 12 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

Proteins are shipped with blue ice.

Protein 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).

Reference

Armes NA, et al. (1997) The ALK-2 and ALK-4 activin receptors transduce distinct mesoderm-inducing signals during early Xenopus development but do not co-operate to establish thresholds. Development 124(19): 3797-804.

Armes NA, et al. (1999) A short loop on the ALK-2 and ALK-4 activin receptors regulates signaling specificity but cannot account for all their effects on early Xenopus development. J Biol Chem. 274(12):7929-35.

Kawai S, et al. (2000) Mouse smad8 phosphorylation downstream of BMP receptors ALK-2, ALK-3, and ALK-6 induces its association with Smad4 and transcriptional activity.Biochem Biophys Res Commun. 271(3):682-7.

Deng Y, et al. (2009) Efficient highly selective synthesis of methyl 2-(ethynyl)alk-2(E)-enoates and 2-(1'-chlorovinyl)alk-2(Z)-enoates from 2-(methoxycarbonyl)-2,3-allenols. Organic letters 11(10):2169-72.