Data Sheet (Cat.No.TMPC-00011)

TargetMol

ALK-2/ACVR1 Protein, Human, Recombinant (G328E, GST)

General Information

Protein Construction:	Recombinant human ALK2 (G328E) (147-end) was expressed by baculovirus in Sf9 insect cells using an N-terminal GST tag.
Species:	Human
Expression Host:	Baculovirus-Insect Cells
Accession:	Q04771
Molecular Weight:	~67 kDa

QC Testing

Biological Activity:	The specific activity of ALK2 (G328E) was determined to be 9 nmol /min/mg by radioactive kinase assay.
Purity:	>90% as determined by SDS-PAGE.
Formulation:	Supplied as sterile 50 mM Tris-HCl, pH 7.5, 150 mM NaCl, 10 mM glutathione, 0.1 mM EDTA, 0.25 mM DTT, 0.1 mM PMSF, 25% glycerol.

Preparation and Storage

Stability & Storage:

Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

Enzymes are highly recommended to be shipped at frozen temperature with dry ice. Shipment made at ambient temperature may seriously affect the activity of the ordered products.

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).

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