Data Sheet (Cat.No.TMPY-04366)

TargetMol

ALK-5 Protein, Human, Recombinant (aa 200-503, His & GST)

General Information

Synonyms:	LDS1;ALK-5;LDS2A;AAT5;SKR4;transforming growth factor, beta receptor 1;tbetaR-I;ESS1; tβR-I;TGFR-1;ALK5;MSSE;ACVRLK4;transforming growth factor, β receptor 1;LDS1A
Protein Construction:	A DNA sequence encoding the human ALK5 (P36897-1) (Thr200-Mey503) was fused with the N-terminal polyhistidine-tagged GST tag at the N-terminus. Predicted N terminal: Met
Species:	Human
Expression Host:	Baculovirus Insect Cells
Accession:	P36897-1
Molecular Weight:	62.6 kDa (predicted); 57 kDa (reducing conditions)

QC Testing

Biological Activity:	The specific activity was determined to be > 5 nmol/min/mg using casein as substrate.
Purity:	> 90 % 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 8.5, 10% glycerol.

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

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

Transforming growth factor, beta receptor I, also known as Transforming growth factor-beta receptor type I , Serine / threonine-protein kinase receptor R4, Activin receptor-like kinase 5, SKR4, ALK-5, and TGFBR1, is a single-pass type I membrane protein that belongs to the protein kinase superfamily and TGFB receptor subfamily. TGFBR1 / ALK-5 is found in all tissues examined. It is most abundant in placenta and least abundant in brain and heart. TGF-beta functions as a tumor suppressor by inhibiting the cell cycle in the G1 phase. Administration of TGF-beta is able to protect against mammary tumor development in transgenic mouse models in vivo. Disruption of the TGF-beta/SMAD pathway has been implicated in a variety of human cancers, with the majority of colon and gastric cancers being caused by an inactivating mutation of TGF-beta RII. On ligand binding, TGFBR1 / ALK-5 forms a

receptor complex consisting of two type I I and two type I transmembrane serine/threonine kinases. Type II receptors phosphorylate and activate type I receptors which auto-phosphorylate, then bind and activate SMAD transcriptional regulators. TGF-beta signaling via TGFBR1 / ALK-5 is not required in myocardial cells during mammalian cardiac development, but plays an irreplaceable cell-autonomous role regulating cellular communication, differentiation and proliferation in endocardial and epicardial cells. Defects in TGFBR1 / ALK-5 are the cause of Loeys-Dietz syndrome type 1A (LDS1A), Loeys-Dietz syndrome type 2A (LDS2A), and aortic aneurysm familial thoracic type 5 (AAT5).

Reference

Seki T,et al.(2006) Nonoverlapping expression patterns of ALK1 and ALK5 reveal distinct roles of each receptor in vascular development. Lab Invest. 86(2): 116-29.

Piek E,et al.(1999) TGF-(beta) type I receptor/ALK-5 and Smad proteins mediate epithelial to mesenchymal transdifferentiation in NMuMG breast epithelial cells. J Cell Sci. 112 (24): 4557-68.

Dudas M,et al.(2004) Tgf-beta3-induced palatal fusion is mediated by Alk-5/Smad pathway. Dev Biol. 266(1): 96-108.

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