

Human TGFBR1 / ALK-5 / SKR4 Protein (aa 200-503, His & GST Tag)

Catalog Number: 10459-H20B



Sino Biological
Biological Solution Specialist

General Information

Gene Name Synonym:

AAT5; ACVRLK4; ALK-5; ALK5; ESS1; LDS1; LDS1A; LDS2A; MSSE; SKR4; tbetaR-I; TGFR-1

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.

Source: Human

Expression Host: Baculovirus-Insect Cells

QC Testing

Purity: > 90 % as determined by SDS-PAGE

Bio Activity:

The specific activity was determined to be > 5 nmol/min/mg using casein as substrate.

Endotoxin:

< 1.0 EU per µg of the protein as determined by the LAL method

Stability:

Samples are stable for up to twelve months from date of receipt at -70 °C

Predicted N terminal: Met

Molecular Mass:

The recombinant human ALK5/GST chimera consists of 541 amino acids and has a calculated molecular mass of 62.6 kDa. The recombinant protein migrates approximately 57 kDa band in SDS-PAGE under reducing conditions.

Formulation:

Supplied as sterile 20mM Tris, 500mM NaCl, pH 8.5, 10% glycerol

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Storage:

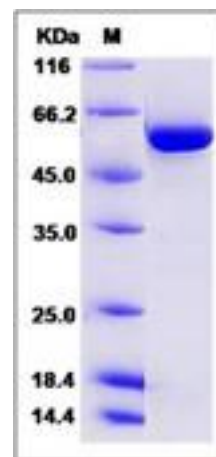
Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:

Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:



Protein Description

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 which belongs to the protein kinase superfamily and TGF-beta 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 II 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).

References

1. 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. *et al.*
2. 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. *et al.*
3. 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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