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

Catalog Number:PKSH030413

Note: Centrifuge before opening to ensure complete recovery of vial contents.

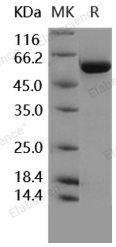
Description

Synonyms	AAT5;ACVRLK4;ALK-5;ALK5;ESS1;LDS1;LDS1A;LDS2A;MSSE;SKR4;tbetaR-I;TGFR-1
Species	Human
Expression Host	Baculovirus-Insect Cells
Sequence	Thr 200-Mey503
Accession	P36897-1
Calculated Molecular Weight	62.6 kDa
Observed molecular weight	57 kDa
Tag	N-His-GST
Bioactivity	The specific activity was determined to be 40 nmol/min/mg using casein as substrate.

Properties

Purity	> 95 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Store at < -20°C, stable for 6 months. Please minimize freeze-thaw cycles.
Shipping	This product is provided as liquid. It is shipped at frozen temperature with blue ice/gel packs. Upon receipt, store it immediately at < - 20°C.
Formulation	Supplied as sterile solution of 20mM Tris, 500mM Nacl, pH 8.5, 10% glycerol
Reconstitution	Not Applicable

Data



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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 which 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

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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).

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