

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.

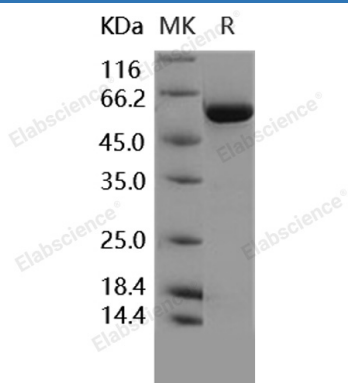
Description

| | |
|----------------------|--|
| Species | Human |
| Source | Baculovirus-Insect Cells-derived Human TGFBR1/ALK-5 protein Thr 200-Mey503, with an N-terminal His & GST |
| Calculated MW | 62.6 kDa |
| Observed MW | 57 kDa |
| Accession | P36897-1 |
| Bio-activity | The specific activity was determined to be 40 nmol/min/mg using casein as substrate. |

Properties

| | |
|----------------------|---|
| Purity | > 95 % as determined by reducing SDS-PAGE. |
| Concentration | Subject to label value. |
| 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 |

Data



> 95 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

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Rev. V3.5

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 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).