Recombinant Human TGFBR1/ALK-5 Protein (His & Fc Tag)

Catalog No. PKSH031596

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

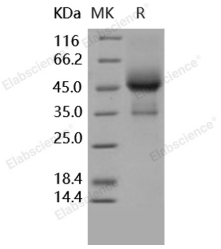
Description

Synonyms	AAT5;ACVRLK4;ALK-5;ALK5;ESS1;LDS1;LDS1A;LDS2A;MSSE;SKR4;tbetaR-I;TGFR-1
Species	Human
Expression Host	HEK293 Cells
Sequence	Met 1-Glu 125
Accession	NP_004603.1
Calculated Molecular Weight	38.8 kDa
Observed molecular weight	45-50 kDa
Tag	C-His-Fc
Bioactivity	Immobilized mouse CD105 at 10 μg/ml (100 μl/well) can bind human TGFRB1 with a linear ranger of 6. 4-800 ng/ml.

Properties

Purity	> 85 % as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU per μg of the protein as determined by the LAL method.
Storage	Generally, lyophilized proteins are stable for up to 12 months when stored at -20 to -80℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ for 3 months.
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.
Formulation	Lyophilized from sterile PBS, pH 7.4 Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Please refer to the specific buffer information in the printed manual.
Reconstitution	Please refer to the printed manual for detailed information.

Data



> 85 % as determined by reducing SDS-PAGE.

Background

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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 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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