



Recombinant Protein Technical Manual

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

RPES3017

Product Data:

Product SKU: RPES3017

Size: 10µg

Species: Human

Expression host: HEK293 Cells

Uniprot: NP_004603.1

Protein Information:

Molecular Mass: 38.8 kDa

AP Molecular Mass: 45-50 kDa

Tag: C-His & Fc

Bio-activity: 1. Measured by its binding ability in a functional ELISA. Immobilized mouse CD105 at 10 µg/ml (100 µl/well) can bind human TGFBR1 with a linear ranager of 6.4-800 ng/ml.2. Measured by its ability to bind human CD105 in a functional ELISA.

Purity: > 85 % as determined by reducing SDS-PAGE.

Endotoxin: < 1.0 EU per µg as determined by the LAL method.

Storage: Lyophilized proteins are stable for up to 12 months when stored at -20 to -80°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of reconstituted samples are stable at < -20°C 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

Reconstitution: Please refer to the printed manual for detailed information.

Application: Functional ELISA

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

Immunogen Information:

Sequence: Met 1-Glu 125

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 TGFBR 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).