Recombinant Protein Technical Manual

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

RPES1889

Product Data:

Product SKU: RPES1889

Size: 20µg

Species: Human

Expression host: Baculovirus-Insect Cells

Uniprot: P36897

Protein Information:

Molecular Mass:	62.6 kDa
AP Molecular Mass:	57 kDa
Tag:	N-His & GST
Bio-activity:	The specific activity was determined to be 40 nmol/min/mg using casein as substrate.
Purity:	> 95 % as determined by reducing SDS-PAGE.
Endotoxin:	< 1.0 EU per µg 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 20mM Tris, 500mM Nacl, pH 8.5, 10% glycerol
Reconstitution:	Please refer to the printed manual for detailed information.
Application:	
Synonyms:	AAT5;ACVRLK4;ALK-5;ALK5;ESS1;LDS1;LDS1A;LDS2A;MSSE;SKR4;tbetaR-I;TGFR

Immunogen Information:

Sequence: Thr 200-Mey503

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 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).