Recombinant Rat TGFBR1 protein (His Tag)

Catalog Number: PDER100218



Description			
Species	Rat		
Mol_Mass	20.9 kDa		
Accession	P80204		
Bio-activity	Not validated for activity		
Properties			
Purity	> 95% as determined by reducing SDS-PAGE.		
Endotoxin	< 10 EU/mg 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		
	°C. Reconstituted protein solution can be stored at 4-8°C for 2-7 days. Aliquots of		
	reconstituted samples are stable at $< -20^{\circ}$ C for 3 months.		
Shipping	This product is provided as lyophilized powder which is shipped with ice packs.		
Formulation	Lyophilized from a 0.2 μ m filtered solution in PBS with 5% Trehalose and 5%		
	Mannitol.		
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of		
	0.5 mg/mL. Concentration is measured by UV-Vis.		

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

KDa	М	R
80		
60		
40		
30		
30		
20		
10		
12		

> 95 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

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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 that 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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