

## Recombinant Rat TGFBR1 Protein (His Tag)

**Catalog Number:** PDER100218

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

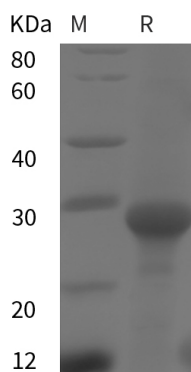
### Description

<b>Species</b>	Rat
<b>Source</b>	E.coli-derived Rat TGFBR1 protein Leu311-Met501, with an N-terminal His
<b>Calculated MW</b>	20.9 kDa
<b>Observed MW</b>	28 kDa
<b>Accession</b>	P80204
<b>Bio-activity</b>	Not validated for activity

### Properties

<b>Purity</b>	> 95% as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	< 10 EU/mg of the protein as determined by the LAL method
<b>Storage</b>	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°C for 3 months.
<b>Shipping</b>	This product is provided as lyophilized powder which is shipped with ice packs.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
<b>Reconstitution</b>	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.

### Data



SDS-PAGE analysis of Rat TGFBR1 proteins, 2 µg/lane of Recombinant Rat TGFBR1 proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 28 kDa.

### Background

#### For Research Use Only

Toll-free: 1-888-852-8623  
Web: [www.elabscience.com](http://www.elabscience.com)

Tel: 1-832-243-6086  
Email: [techsupport@elabscience.com](mailto:techsupport@elabscience.com)

Fax: 1-832-243-6017

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 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).

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