

## Recombinant Mouse ALK-5/TGFBR1 (C-Fc)

**Catalog Number:** PKSM041408

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

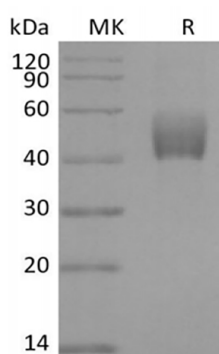
### Description

<b>Species</b>	Mouse
<b>Source</b>	HEK293 Cells-derived Mouse ALK-5/TGFBR1 protein Leu30-Glu125, with an C-terminal Fc
<b>Calculated MW</b>	37.6 kDa
<b>Observed MW</b>	40-60 kDa
<b>Accession</b>	Q64729
<b>Bio-activity</b>	Not validated for activity

### Properties

<b>Purity</b>	> 95 % as determined by reducing SDS-PAGE.
<b>Endotoxin</b>	< 1.0 EU per µg 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 of PBS, pH 7.4. Normally 5% - 8% trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
<b>Reconstitution</b>	Please refer to the specific buffer information in the printed manual. Please refer to the printed manual for detailed information.

### Data



> 95 % as determined by reducing SDS-PAGE.

### Background

### For Research Use Only

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TGF-beta RI, also called ALK-5, is an approximately 55 kDa type I transmembrane serine/threonine receptor kinase. In the presence of TGF-beta, TGF-beta RI forms a complex with, and is phosphorylated by, TGF-beta RII. Phosphorylated TGF-beta RI can then transiently bind and phosphorylate Smad2 and Smad3. 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. TGF-beta RI is likely important during development, since mice deficient for TGF-beta RI die at midgestation with severe defects in vascular development of the yolk sac and placenta, and an absence of circulating red blood cells. Furthermore, TGF-beta RI appears to be involved in proper lymphatic network development.