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

Catalog No. PKSM041408

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

## **Description**

**Synonyms** AAT5;activin A receptor type II-like kinase;53kD;ACVRLK4;ALK-5;ALK-5ALK5

;LDS1A;LDS2A;SKR4;tbetaR-I;TGFB1R1;TGF-beta receptor type

I;TGFbetaRI;TGFBR1;TGF-bRI;TGFR-1

**Species** Mouse

HEK293 Cells **Expression Host** Leu30-Glu125 Sequence Q64729 Accession Calculated Molecular Weight 37.6 kDa Observed molecular weight 40-60 kDa C-Fc Tag

#### **Properties**

**Purity** > 95 % as determined by reducing SDS-PAGE.

**Endotoxin** < 1.0 EU per µg 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°C for 3 months.

**Shipping** This product is provided as lyophilized powder which is shipped with ice packs.

**Formulation** Lyophilized from a 0.2 µm filtered solution of PBS, pH 7.4.

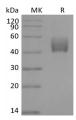
Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as

protectants before lyophilization.

Please refer to the specific buffer information in the printed manual.

Reconstitution Please refer to the printed manual for detailed information.

## Data



> 95 % as determined by reducing SDS-PAGE.

# **Background**

TGF-beta RI, also called ALK-5, is an approximately 55 kDa type I transmembrane serine/threonine receptor kinase. In

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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 TGFbeta 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.

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