Recombinant Human ALK-5/TGFBR1 (C-Fc)

Catalog Number:PKSH033907

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	Human
Expression Host	HEK293 Cells
Sequence	Leu34-Glu125
Accession	P36897
Calculated Molecular Weight	37.2 kDa
Observed molecular weight	40-60 kDa
Tag	C-Fc

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℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ 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.

Background

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.

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