TGFBR1/ALK-5 Protein, Human (HEK293, His-Fc)

Cat. No.:	HY-P74523
Synonyms:	TGF-beta receptor type-1; ALK-5; SKR4; TbetaR-I; AAT5
Species:	Human
Source:	HEK293
Accession:	P36897-1 (L34-E125)
Gene ID:	7046
Molecular Weight:	45-50 kDa

PROPERTIES

Biological Activity	The enzyme activity of this recombinant protein is testing in progress, we cannot offer a guarantee yet.
Appearance	Lyophilized powder.
Formulation	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.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US; may vary elsewhere.

DESCRIPTION

Background	The transmembrane serine/threonine kinase, TGFBR1 (ALK-5), collaborates with the TGF-beta type II serine/threonine kinase receptor, TGFBR2, to form the dedicated receptor for TGF-beta cytokines, including TGFB1, TGFB2, and TGFB3. Serving as a signal transducer, TGFBR1 mediates the transmission of TGFB1, TGFB2, and TGFB3 signals from the cell surface to the cytoplasm, thereby orchestrating a diverse array of physiological and pathological processes. These include cell cycle arrest in epithelial and hematopoietic cells, control of mesenchymal cell proliferation and differentiation, wound healing, extracellular matrix production, immunosuppression, and carcinogenesis. The receptor complex, composed of 2 TGFBR1 and 2 TGFBR2 molecules symmetrically bound to the cytokine dimer, leads to the phosphorylation and activation of TGFBR1 by the constitutively active TGFBR2. Activated TGFBR1 phosphorylates SMAD2, causing its dissociation from the receptor and interaction with SMAD4. The resulting SMAD2-SMAD4 complex translocates to the nucleus, where it modulates the transcription of TGF-beta-regulated genes, constituting the canonical SMAD-dependent TGF-beta signaling cascade. Additionally, TGFBR1 is involved in non-canonical, SMAD-independent TGF-beta signaling pathways. For instance, it induces TRAF6 autoubiquitination, leading to MAP3K7 ubiquitination and activation, triggering apoptosis. TGFBR1 also regulates epithelial to mesenchymal transition through a SMAD-independent signaling pathway involving PARD6A phosphorylation

	and activation.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA