TGFBR1/ALK-5 Protein, Human (sf9, His-GST)

Cat. No.:	HY-P74522
Synonyms:	TGF-beta receptor type-1; ALK-5; SKR4; TbetaR-I; AAT5
Species:	Human
Source:	Sf9 insect cells
Accession:	P36897 (T200-M503)
Gene ID:	7046
Molecular Weight:	Approximately 57 kDa

PROPERTIES

Biological Activity	The specific activity was determined to be > 5 nmol/min/mg using casein as substrate.
Appearance	Solution
Formulation	Supplied as a 0.2 μm filtered solution of 20 mM Tris, 500 mM NaCl, pH 8.5, 10% glycerol.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	N/A.
Storage & Stability	Stored at -80°C for 1 year. It is stable at -20°C for 3 months after opening. It is recommended to freeze aliquots at -80°C for extended storage. Avoid repeated freeze-thaw cycles.
Shipping	Shipping with dry ice

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.

Caution: Product has not been fully validated for medical applications. For research use only.

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