Human alk5 / TGFBR1 Protein (ECD, Fc Tag)

Catalog Number: 10459-H02H

General Information

Gene Name Synonym:
AAT5; ACVRLK4; ALK-5; ALK5; ESS1; LDS1; LDS1A; LDS2A; MSSE; SKR4; tbetaR-I; TGFR-1

Protein Construction:
A DNA sequence encoding the human TGFBR1 isoform 1 precursor (NP_004603.1) (Met1-Leu126) was expressed with the Fc region of human IgG1 at the C-terminus.

Source: Human
Expression Host: HEK293 Cells

QC Testing

Purity: > 95 % as determined by SDS-PAGE.

Endotoxin:
< 1.0 EU per µg protein as determined by the LAL method.

Predicted N terminal: Leu34

Molecular Mass:
The recombinant human TGFBR1 consists of 331 amino acids and predicts a molecular mass of 36.87 kDa.

Formulation:
Lyophilized from sterile PBS, pH 7.4

Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as protectants before lyophilization. Specific concentrations are included in the hardcopy of COA. Please contact us for any concerns or special requirements.

Usage Guide

Stability & Storage:
Samples are stable for twelve months from date of receipt at -20°C to -80°C.

Store it under sterile conditions at -20°C to -80°C upon receiving. Recommend to aliquot the protein into smaller quantities for optimal storage.

Avoid repeated freeze-thaw cycles.

Reconstitution:
Detailed reconstitution instructions are sent along with the products.

SDS-PAGE:

Protein Description

Transforming growth factor, beta receptor I, also known as Transforming growth factor-beta receptor type I, Serine / threonine-protein kinase receptor R4, Activin receptor-like kinase 5, SKR4, ALK-5, and TGFBR1, is a single-pass type I membrane protein which belongs to the protein kinase superfamily and TGFBR receptor subfamily. TGFBR1 / ALK-5 is found in all tissues examined. It is most abundant in placenta and least abundant in brain and heart. 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. On ligand binding, TGFBR1 / ALK-5 forms a receptor complex consisting of two type I I and two type I transmembrane serine/threonine kinases. Type II receptors phosphorylate and activate type I receptors which auto-phosphorylate, then bind and activate SMAD transcriptional regulators. TGF-beta signaling via TGFBR1 / ALK-5 is not required in myocardial cells during mammalian cardiac development, but plays an irreplaceable cell-autonomous role regulating cellular communication, differentiation and proliferation in endocardial and epicardial cells. Defects in TGFBR1 / ALK-5 are the cause of Loeys-Dietz syndrome type 1A (LDS1A), Loeys-Dietz syndrome type 2A (LDS2A), and aortic aneurysm familial thoracic type 5 (AAT5).

References