Recombinant Human TMED1 (C-Fc)

Catalog Number: PKSH033904

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

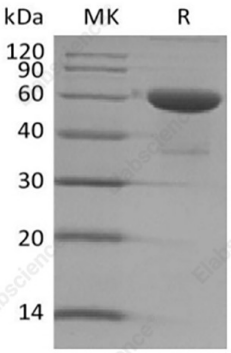
Description

Species	Human
Source	HEK293 Cells-derived Human TMED1 protein Ala24-Asn194, with an C-terminal Fc
Calculated MW	46.3 kDa
Observed MW	55-65 kDa
Accession	Q13445
Bio-activity	Not validated for activity

Properties

Purity	> 90 % 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% Tween 80 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



> 90 % as determined by reducing SDS-PAGE.

Background

TMED1 (Transmembrane Emp24 domain-containing protein 1) is a member of the TMED family of proteins (gene name TMED1). The TMED family of proteins are localized to membranes of the early secretory pathway, including the endoplasmic reticulum and Golgi, and function in vesicular protein trafficking. TMED1 is a 59 kDa monomer and has been reported to exist as homodimer. It contains 1 GOLD domain and is widely expressed. TMED1 is important in regulating innate immune signaling through its interaction with ST2L. Specifically, the GOLD domain in TMED1 interacts with the TIR domain of ST2L, a receptor for IL 33. This interaction promotes ST2L association with IL-33, allowing downstream signaling cascade activating MAP kinases, p38, and JNK. Studies have shown knockdown of TMED-1 in HUVECs impairs the IL-33 induced response resulting in reduction of IL-6 and IL-8 productions.

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