

Recombinant Human STIM1/GOK Protein (His Tag)

Catalog Number: PKSH031120

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

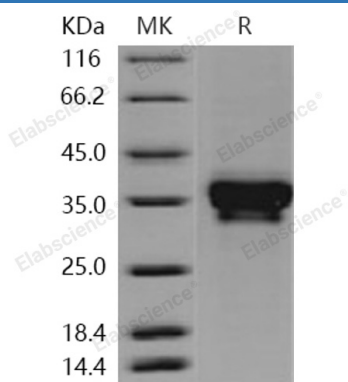
Description

Species	Human
Source	HEK293 Cells-derived Human STIM1/GOK protein Met 1-Asp 213, with an C-terminal His
Calculated MW	23.3 kDa
Observed MW	33-38 kDa
Accession	NP_003147.2
Bio-activity	Not validated for activity

Properties

Purity	> 97 % 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 sterile 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



> 97 % as determined by reducing SDS-PAGE.

Background

For Research Use Only

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Stromal interaction molecule 1, also known as STIM1 and GOK, is a cell membrane, a single-pass type I membrane protein and an endoplasmic reticulum membrane protein. STIM1 / GOK is ubiquitously expressed in various human primary cells and tumor cell lines. It contains one EF-hand domain and one SAM (sterile alpha motif) domain. STIM1 / GOK plays a role in mediating Ca²⁺ influx following depletion of intracellular Ca²⁺ stores. It acts as Ca²⁺ sensor in the endoplasmic reticulum via its EF-hand domain. Upon Ca²⁺ depletion, STIM1 / GOK translocates from the endoplasmic reticulum to the plasma membrane where it activates the Ca²⁺ release-activated Ca²⁺ (CRAC) channel subunit, TMEM142A / ORAI1. Transfection of STIM1 / GOK into cells derived from a rhabdoid tumor and from a rhabdomyosarcoma that do not express detectable levels of STIM1 can induce cell death, suggesting a possible role in the control of rhabdomyosarcomas and rhabdoid tumors. Defects in STIM1 are the cause of immune dysfunction with T-cell inactivation due to calcium entry defect type 2 (IDTICED2) which is an immune disorder characterized by recurrent infections, impaired T-cell activation and proliferative response, decreased T-cell production of cytokines, lymphadenopathy, and normal lymphocyte counts and serum immunoglobulin levels.