Recombinant Human STIM1/GOK Protein (His Tag)

Catalog No. PKSH031120

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

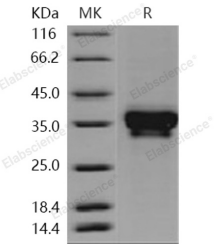
Description

Synonyms	D11S4896E;GOK;IMD10;STRMK;TAM;TAM1
Species	Human
Expression Host	HEK293 Cells
Sequence	Met 1-Asp 213
Accession	NP_003147.2
Calculated Molecular Weight	23.3 kDa
Observed molecular weight	33-38 kDa
Tag	C-His
Bioactivity	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℃. Reconstituted protein solution can be stored at 4-8℃ for 2-7 days. Aliquots of reconstituted samples are stable at < -20℃ 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% Tween80 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

Stromal interaction molecule 1, also known as STIM1 and GOK, is a cell membrane, a single-pass type I membrane protein and a endoplasmic reticulum membrane protein. STIM1 / GOK is ubiquitously expressed in various human

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primary cells and tumor cell lines. It contains oneEF-hand domain and oneSAM (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 lymphocytes counts and serum immunoglobulin levels.

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