## Recombinant Human STIM1/GOK Protein (His Tag)

Catalog Number: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

#### **Properties**

**Purity** > 97 % as determined by reducing SDS-PAGE.

**Endotoxin**  $< 1.0 \text{ EU per } \mu \text{g of the protein as determined by the LAL method.}$ 

**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

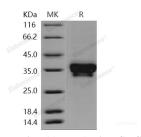
Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 are added as

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 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

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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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