Recombinant Human TREM1 Protein (His &Fc Tag)

Catalog Number: PKSH031561

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

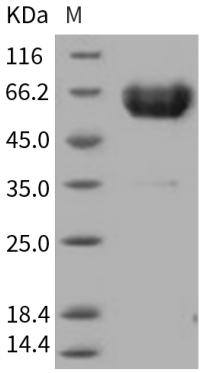
Description

Species	Human
Source	HEK293 Cells-derived Human TREM1 protein Met 1-Arg 200, with an C-terminal His & Fc
Calculated MW	48.3 kDa
Observed MW	60-65 kDa
Accession	NP_061113.1
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

TREM1 (triggering receptor expressed on myeloid cells) is a type I transmembrane protein with a single Ig-like domain; and is selectively expressed on blood neutrophils and a subset of monocytes. As a member of the growing family of receptors related to NK cell receptors; TREM1 activates downstream signaling events with the help of an adapter protein called DAP12. Expression of TREM1 is up-regulated by bacterial LPS; a ligand for TLR4; as well as lipoteichoic acid. Although its natural ligand has not been identified; engagement of TREM1 with agonist mAbs triggers secretion of the proinflammatory cytokines TNF-α and IL-1β; as well as chemokines such as IL-8 and monocyte chemoattractant protein (MCP)-1. Intracellularly; TREM1 induces Ca2+ mobilization and tyrosine phosphorylation of extracellular signal-related kinase 1 (ERK1); ERK2 and phospholipase C-γ. In an animal model of LPS-induced septic shock; blockade of TREM1 signaling inhibited hyperresponsiveness and death. Thus; it has been demonstrated that TREM1 performs a critical function in immune responses involved in host defense against microbial challenges; and is suggested to be a potential therapeutic target for septic shock.

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