Recombinant Human TREM-1 Protein(His Tag)

Catalog Number: PDMH100168

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

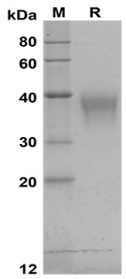
Description

Species	Human
Source	Mammalian-derived Human TREM-1 protein Met1-Arg200, with an C-terminal His
Calculated MW	21.9 kDa
Observed MW	30-40 kDa
Accession	Q9NP99
Bio-activity	Not validated for activity

Properties

Purity	> 95% as determined by reducing SDS-PAGE.
Endotoxin	< 1.0 EU/mg 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 a 0.2 μm filtered solution in PBS with 5% Trehalose and 5% Mannitol.
Reconstitution	It is recommended that sterile water be added to the vial to prepare a stock solution of 0.5 mg/mL. Concentration is measured by UV-Vis.

Data



SDS-PAGE analysis of Human TREM-1 proteins, 2μg/lane of Recombinant Human TREM-1 proteins was resolved with SDS-PAGE under reducing conditions, showing bands at 30-40 kDa

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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