

TREM-1 Protein, Mouse, Recombinant (His)

General Information

Synonyms:	triggering receptor expressed on myeloid cells 1
Protein Construction:	A DNA sequence encoding the mouse TREM1 (Q9JKE2-1) (Met1-Ser202) was expressed with a polyhistidine tag at the C-terminus. Predicted N terminal: Ala 21
Species:	Mouse
Expression Host:	HEK293 Cells
Accession:	Q9JKE2-1
Molecular Weight:	21.57 kDa (predicted); 41.55 kDa (reducing conditions)

QC Testing

Biological Activity:	Activity testing is in progress. It is theoretically active, but we cannot guarantee it. If you require protein activity, we recommend choosing the eukaryotic expression version first.
Purity:	> 95 % as determined by SDS-PAGE.
Endotoxin:	< 1.0 EU/μg of the protein as determined by the LAL method.
Formulation:	Lyophilized from a solution filtered through a 0.22 μm filter, containing PBS, pH 7.4. Typically, a mixture containing 5% to 8% trehalose, mannitol, and 0.01% Tween 80 is incorporated as a protective agent before lyophilization.

Preparation and Storage

Reconstitution:
A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

It is recommended to store recombinant proteins at -20°C to -80°C for future use. Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry ice.

Protein 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-1 β , 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.

Reference

Bouchon, A. et al., 2000, J. Immunol. 164: 4991-4995.

Bouchon, A. et al., 2001, Nature. 410: 1103-1107.

Bleharski, J.R. et al., 2003, J. Immunol. 170: 3812-3818.

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