TREM-1 Protein, Mouse (HEK293, His)

Cat. No.:	HY-P70716
Synonyms:	Triggering receptor expressed on myeloid cells 1; TREM-1; CD354; Trem1
Species:	Mouse
Source:	HEK293
Accession:	Q9JKE2 (A21-S202)
Gene ID:	58217
Molecular Weight:	Approximately 38.0 kDa

PROPERTIES

AA Sequence	A I V L E E E R Y D L V E G Q T L T V K C P F N I M K Y A N S Q K A W Q R L P D G K E P L T L V V T Q R P F T R P S E V H M G K F T L K H D P S E A M L Q V Q M T D L Q V T D S G L Y R C V I Y H P P N D P V V L F H P V R L V V T K G S S D V F T P V I I P I T R L T E R P I L I T T K Y S P S D T T T T R S L P K P T A V V S S P G L G V T I I N G T D A D S V S T S S
Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm filtered solution of PBS, pH 7.4.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH_2O. For long term storage it is recommended to add a carrier protein (0.1% BSA, 5% HSA, 10% FBS or 5% Trehalose).
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.
Shipping	Room temperature in continental US;may vary elsewhere.

DESCRIPTION

Background	TREM-1, a cell surface receptor, assumes pivotal roles in both innate and adaptive immunity by robustly amplifying inflammatory responses. Upon activation by diverse ligands such as PGLYRP1, HMGB1, or HSP70, TREM-1 undergoes multimerization and forms a complex with the transmembrane adapter TYROBP/DAP12. This initiates a SYK-mediated cascade of tyrosine phosphorylation, activating downstream mediators like BTK, MAPK1, MAPK3, or phospholipase C-gamma. Consequently, this cascade facilitates the release of pro-inflammatory cytokines and/or chemokines by neutrophils and macrophages, promoting their migration and thereby amplifying inflammatory responses triggered by bacterial and fungal infections. Beyond microbial interactions, TREM-1 also contributes to the amplification of inflammatory signals

	initiated by Toll-like receptor (TLR) and NOD-like receptor engagement, playing a crucial role in the pathophysiology of various acute and chronic inflammatory diseases, including septic shock and atherosclerosis. In its monomeric state, TREM-1 forms homomultimers upon activation and interacts with TYROBP/DAP12 and TLR4, further highlighting its intricate involvement in the regulation of immune responses.

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