Recombinant Mouse TREM-2

Catalog Number: 9228-T2

DESCRIPTION

Source	Mouse myeloma cell line, NS0-derived Leu19-Ser171, with a C-terminal 6-His tag Accession # Q99NH8
N-terminal Sequence Analysis	Leu19
Predicted Molecular Mass	18 kDa

SPECIFICATIONS

Activity	Measured by its ability to bind fluorescein-conjugated *S. aureus* Bioparticles. Daws, M.R. *et al.* (2003) J. Immunol. **171**:594. The ED_{50} for this effect is 20-120 ng/mL using a His tag antibody coated plate.
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in HEPES and NaCl. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/mL in water.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 3 months, -20 to -70 °C under sterile conditions after reconstitution.

BACKGROUND

TREM-2 (Triggering Receptor Expressed on Myeloid cells-2) is a 35 kDa type I transmembrane member of the TREM family and Ig superfamily (1). Mature mouse TREM-2 consists of a 153 amino acid (aa) extracellular domain (ECD) with one Ig-like domain, a 21 aa transmembrane segment, and a 35 aa cytoplasmic domain (2). Within the ECD, mouse TREM-2 shares 73% and 90% aa sequence identity with human and rat TREM-2, respectively. Soluble forms of the TREM-2 ECD are generated by alternative splicing or proteolytic cleavage, and the cytoplasmic domain can be liberated by gamma-Secretase mediated intramembrane cleavage (3). A positively charged lysine within the transmembrane segment allows association with the signal adapter protein, DAP12 and inhibition of macrophage activation (4, 5). TREM-2 is expressed on macrophages, immature myeloid dendritic cells, osteoclasts, microglia, and adipocytes (5-9). It promotes the differentiation and function of osteoclasts, the production of inflammatory cytokines by adipocytes, insulin resistance, and the phagocytic clearance of bacteria (9-11). In the CNS, TREM-2 binds to ApoE, ApoA1, and ApoB and mediates the clearance of apoptotic neurons, amyloid plaques, and cell debris following demyelination (6-8, 12). TREM-2 also interacts with and modifies signaling through Plexin A1 on dendritic cells and osteoclasts (13). Mutations in TREM-2 or DAP12 are associated with the development of Alzheimer's disease and Nasu-Hakola disease (NHD/PLOSL) which is characterized by presenile dementia and bone cysts (14, 15). Soluble TREM-2 is elevated in cerebrospinal fluid of patients with active multiple sclerosis (MS), and TREM-2 blockade exacerbates disease symptoms in the experimental EAE model of MS (16, 17).

References:

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