Human TREM2 Antibody

Monoclonal Mouse IgG_1 Clone # 1057114

Catalog Number: MAB18282

DESCRIPTION

Species Reactivity	Human
Specificity	Detects Human Trem-2 in direct and capture ELISAs
Source	Monoclonal Mouse IgG_1 Clone # 1057114
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	NS0-derived recombinant human TREM-2 protein His19-Ser174
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 μm filtered solution in PBS.

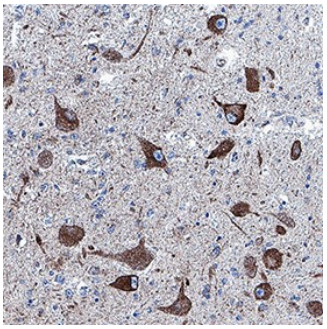
APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

	Recommended Concentration	Sample
Immunohistochemistry	5-25 µg/mL	Immersion fixed paraffin-embedded sections of Human Brain Medulla (Locus Coeruleus)

DATA

Immunohistochemistry



Detection of TREM2 in Human Brain Medulla (Locus Coeruleus). TREM2 was detected in immersion fixed paraffin-embedded sections of Human Brain Medulla (Locus Coeruleus) using Mouse Anti-Human TREM2 Monoclonal Antibody (Catalog # MAB18282) at 5 µg/mL for 1 hour at room temperature followed by incubation with the Anti-Mouse IgG VisUCyte™ HRP Polymer Antibody (Catalog # VC001). Before incubation with the primary antibody, tissue was subjected to heat-induced epitope retrieval using VisUCyte Antigen Retrieval Reagent-Basic (Catalog # VCTS021). Tissue was stained using DAB (brown) and counterstained with hematoxylin (blue). Specific staining was localized to cytoplasm in neurons. View our protocol for IHC Staining with VisUCyte HRP Polymer Detection Reagents.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
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. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

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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 human TREM-2 consists of a 156 amino acid (aa) extracellular domain (ECD) with one V-type Ig-like domain, a 21 aa transmembrane (TM) domain, and a 35 aa cytoplasmic tail (2). Within the ECD, human TREM-2 shares 73% and 74% aa sequence identity with mouse 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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