Human TREM2 Antibody

Recombinant Monoclonal Rabbit IgG Clone # 2958E
Catalog Number: MAB11618

DESCRIPTION

Species Reactivity	Human
Specificity	Detects recombinant human TREM2 protein in Direct ELISA.
Source	Recombinant Monoclonal Rabbit IgG Clone # 2958E
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line, NS0-derived human TREM2 His19-Ser174 Accession # Q9NZC2
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose.

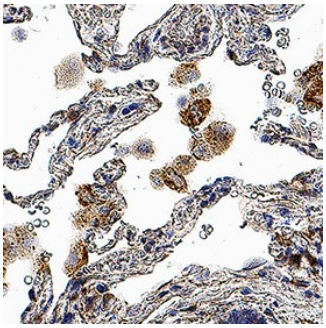
APPLICATIONS

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	1-10 µg/mL	Immersion fixed paraffi-embedded sections of human lung and brain cortex

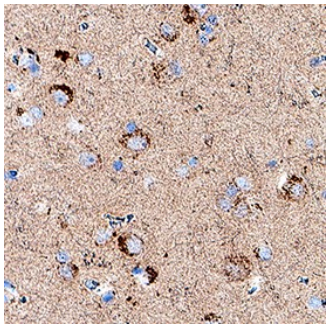
DATA

Immunohistochemistry



Detection of TREM2 in Human Lung. TREM2 was detected in immersion fixed paraffin-embedded sections of human lung using Rabbit Anti-Human TREM2 Monoclonal Antibody (Catalog # MAB11618) at 3 µg/ml for 1 hour at room temperature followed by incubation with the Anti-Rabbit IgG VisUCyte™ HRP Polymer Antibody (Catalog # VC003). 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 the cytoplasm. View our protocol for IHC Staining with VisUCyte HRP Polymer Detection Reagents.

Immunohistochemistry



Detection of TREM2 in Human Brain Cortex. TREM2 was detected in immersion fixed paraffin-embedded sections of human brain cortex using Rabbit Anti-Human TREM2 Monoclonal Antibody (Catalog # MAB11618) at 3 µg/ml for 1 hour at room temperature followed by incubation with the Anti-Rabbit IgG VisUCyte™ HRP Polymer Antibody (Catalog # VC003). 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 the cytoplasm of microglia. View our protocol for IHC Staining with VisUCyte HRP Polymer Detection Reagents.

PREPARATION AND STORAGE

Reconstitution	Reconstitute lyophilized material at 0.2 mg/ml in sterile PBS. For liquid material, refer to CoA for concentration.
Shipping	Lyophilized product is shipped at ambient temperature. Liquid small pack size (-SP) is shipped with polar packs. Upon receipt, store 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. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

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BACKGROUND

TREM2 (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 TREM2 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 TREM2 shares 73% and 74% aa sequence identity with mouse and rat TREM2, respectively. Soluble forms of the TREM2 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). TREM2 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, TREM2 binds to ApoE, ApoA1, and ApoB and mediates the clearance of apoptotic neurons, amyloid plaques, and cell debris following demyelination (6-8, 12). TREM2 also interacts with and modifies signaling through Plexin A1 on dendritic cells and osteoclasts (13). Mutations in TREM2 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 TREM2 is elevated in cerebrospinal fluid of patients with active multiple sclerosis (MS), and TREM2 blockade exacerbates disease symptoms in the experimental EAE model of MS (16, 17).

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