

DESCRIPTION

Species Reactivity	Human
Specificity	Detects recombinant human TREM2 protein in Direct ELISA.
Source	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
Conjugate	Alexa Fluor 594 Excitation Wavelength: 590 nm Emission Wavelength: 617 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide
*Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.	

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.

Immunohistochemistry Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

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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