

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
Conjugate	Alexa Fluor 532 Excitation Wavelength: 534 nm Emission Wavelength: 553 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

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).

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