

Mouse TREM2 Alexa Fluor® 488-conjugated Antibody

Antigen Affinity-purified Polyclonal Sheep IgG Catalog Number: AF1729G

100 µg

DESCRIPTION		
Species Reactivity	Mouse	
Specificity	Detects mouse TREM2 in direct ELISAs and Western blots.	
Source	Polyclonal Sheep IgG	
Purification	Antigen Affinity-purified	
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse TREM2b Leu19-Pro168 Accession # Q99NH8	
Conjugate	Alexa Fluor 488 Excitation Wavelength: 488 nm Emission Wavelength: 515-545 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.		
Knockout Validated	Optimal dilution of this antibody should be experimentally determined.	
Western Blot	Optimal dilution of this antibody should be experimentally determined.	
ELISA	Optimal dilution of this antibody should be experimentally determined.	
Immunocytochemistry	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 by Myeloid cells) is an Ig superfamily cell surface receptor that activates a number of myeloid cell types (1). It is a member of a small gene family located on human chromosome 6p21 and mouse chromosome 17 in a region linked to the MHC (2). A single human TREM2 gene has been described, however, two closely related orthologs were reported in mouse (3). The proteins differ by only three amino acids and were designated TREM2a and TREM2b. TREM2 is type I transmembrane protein consisting of a single extracellular immunoglobulin (V-like) domain, a transmembrane domain with a positively charged lysine residue, and a short cytoplasmic tail (1). It associates with the signal adapter protein, DAP12, for signaling and function. DAP12 has a cytoplasmic ITAM that is phosphorylated upon ligand binding leading to the subsequent activation of cytoplasmic tyrosine kinases. TREM2 is expressed by immature monocyte-derived dendritic cells (DC), and expression is down-regulated upon activation of DC by microbial products and costimulatory signals (4). Ligation of TREM2 on immature DC with anti-TREM2 antibodies results in partial DC activation and the up-regulation of CCR7 and some co-stimulatory molecules. A role for TREM2 in the functioning of osteoclasts and microglia is suggested by the discovery that homozygous loss-of-function mutations in either TREM2 or DAP12 result in Nasu-Hakola disease characterized by a combination of presenile demetia and bone cysts (5). *In vitro* studies indicate that the differentiation of myeloid precursors into osteoclasts is dramatically impaired in TREM2 deficient individuals (6).

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Rev. 9/12/2025 Page 1 of 1

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