Revision 3

Store at -20C

#76765

TREM2 (E7P8J) Rabbit mAb (Carboxy-terminal Antigen)

Cell Signaling TECHNOLOGY®

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Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W	M	Endogenous	11, 28	Rabbit IgG	#Q99NH8	83433

Product Usage Information

Application	Dilution
Western Blotting	1:1000

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

TREM2 (E7P8J) Rabbit mAb (Carboxy-terminal Antigen) recognizes endogenous levels of total mouse TREM2 protein, both the full-length and the carboxy-terminal membrane fragment generated by proteolytic processing. A non-specific band of unknown origin is observed migrating at ~80 kDa.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Gly215 of mouse TREM2 protein.

Background

The triggering receptor expressed on myeloid cells 2 (TREM2) protein is an innate immune receptor that is expressed on the cell surface of microglia, macrophages, osteoclasts, and immature dendritic cells (1). The TREM2 receptor is a single-pass type I membrane glycoprotein that consists of an extracellular immunoglobulin-like domain, a transmembrane domain, and a cytoplasmic tail. TREM2 interacts with the tyrosine kinase-binding protein DAP12 to form a receptor-signaling complex (2). The TREM2 protein plays a role in innate immunity and a rare functional variant (R47H) of TREM2 is associated with the late-onset risk of Alzheimer's disease (1,3). Research studies using mouse models of Alzheimer's disease indicate that deficiency and haploinsufficiency of TREM2 can lead to increased β-amyloid (Aβ) accumulation as a result of dysfunctional microglial response (4). These results agree with the distribution of TREM2 in human brain regions (e.g., white matter, the hippocampus, and neocortex) that are involved in Alzheimer's disease pathology (2). In addition, amyloid plaque formation induces expression of TREM2 and amyloid phagocytosis (5). Loss-of-function mutations in the corresponding *TREM2* or *DAP12* genes can result in Nasu-Hakola disease, a rare form of progressive presenile dementia that results from polycystic osseous lesions (6). TREM2 membrane shedding occurs by cleavage at the extracellular site between H157/S158, generating an N-terminal shedded fragment and a membrane bound C-terminal fragment (7,8).

Background References

1. Colonna, M. (2003) *Nat Rev Immunol* 3, 445-53.
2. Jonsson, T. et al. (2013) *N Engl J Med* 368, 107-16.
3. Boutajangout, A. and Wisniewski, T. (2013) *Int J Cell Biol* 2013, 576383.
4. Wang, Y. et al. (2015) *Cell* 160, 1061-71.
5. Melchior, B. et al. (2010) *ASN Neuro* 2, e00037.
6. Klünemann, H.H. et al. (2005) *Neurology* 64, 1502-7.
7. Thornton, P. et al. (2017) *EMBO Mol Med* 9, 1366-1378.
8. Schlepckow, K. et al. (2017) *EMBO Mol Med* 9, 1356-1365.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting

Cross-Reactivity Key

M: Mouse

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