Mouse M-CSF R Antibody

Antigen Affinity-purified Polyclonal Sheep IgG
Catalog Number: AF3818

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse M-CSF R in direct ELISAs and Western blots. In direct ELISAs, approximately 40% cross-reactivity with recombinant human M-CSF R is observed.
Source	Polyclonal Sheep IgG
Purification	Antigen Affinity-purified
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse M-CSF R Ala20-Ser511 Accession # P09581
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 μm filtered solution in PBS.

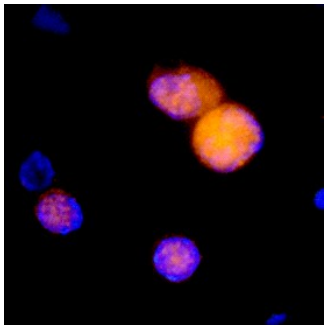
APPLICATIONS

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
Western Blot	0.1 µg/mL	Recombinant Mouse M-CSF R Fc Chimera (Catalog # 3818-MR)
Immunocytochemistry	5-15 µg/mL	See Below

DATA

Immunocytochemistry



M-CSF R in Mouse Splenocytes. M-CSF R was detected in immersion fixed mouse splenocytes using Mouse M-CSF R Antigen Affinity-purified Polyclonal Antibody (Catalog # AF3818) at 15 µg/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Goat IgG Secondary Antibody (orange; Catalog # NL001) and counterstained with DAPI (blue). Specific staining was localized to cytoplasm. View our protocol for Fluorescent ICC Staining of Non-adherent Cells.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
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

M-CSF receptor, the product of the *c-fms* proto-oncogene, is a member of the type III subfamily of receptor tyrosine kinases that also includes receptors for SCF and PDGF. These receptors each contain five immunoglobulin-like domains in their extracellular domain (ECD) and a split kinase domain in their intracellular region (1-4). M-CSF receptor is expressed primarily on cells of the monocyte/macrophage lineage, dendritic cells, stem cells and in the developing placenta (1). Mouse M-CSF receptor cDNA encodes a 977 amino acid (aa) type I membrane protein with a 19 aa signal peptide, a 492 aa extracellular region containing the ligand-binding domain, a 25 aa transmembrane domain and a 441 aa cytoplasmic domain. The mouse M-CSF R ECD shares > 99% aa identity with rat and 60-63% aa identity with corresponding sequences in human, canine, feline and bovine M-CSF R. Activators of protein kinase C induce TACE/ADAM17 cleavage of the M-CSF receptor, releasing the functional ligand-binding extracellular domain (5). M-CSF binding induces receptor homodimerization, resulting in transphosphorylation of specific cytoplasmic tyrosine residues and signal transduction (6). The intracellular domain of activated M-CSF R binds more than 150 proteins that affect cell proliferation, survival, differentiation and cytoskeletal reorganization. Among these, PI3Kinase, P42/44 ERK and c-Cbl are key transducers of M-CSF R signals (3, 4). M-CSF R engagement is continuously required for macrophage survival and regulates lineage decisions and maturation of monocytes, macrophages, osteoclasts and DC (3, 4). M-CSF R and integrin $\alpha_v\beta_3$ share signaling pathways during osteoclastogenesis, and deletion of either causes osteopetrosis (7, 8). In the brain, microglia expressing increased M-CSF R are concentrated with Alzheimers aβ peptide, but their role in pathogenesis is unclear (9, 10).

References:

1. deParseval, N. *et al.* (1993) Nucleic Acids Res. **21**:750.
2. Rothwell, V.M. and L.R. Rohrschneider (1987) Oncogene Res. **1**:311.
3. Chitu, V. and E.R. Stanley (2006) Curr. Opin. Immunol. **18**:39.
4. Ross, F.P. and S.L. Teitelbaum (2005) Immunol. Rev. **208**:88.
5. Rovida, E. *et al.* (2001) J. Immunol. **166**:1583.
6. Yeung, Y. *et al.* (1998) J. Biol. Chem. **273**:17128.
7. Dai, X. *et al.* (2002) Blood **99**:111.
8. Faccio, R. *et al.* (2003) J. Clin. Invest. **111**:749.
9. Li, M. *et al.* (2004) J. Neurochem. **91**:623.
10. Mitrasinovic, O.M. *et al.* (2005) J. Neurosci. **25**:4442.