

DESCRIPTION

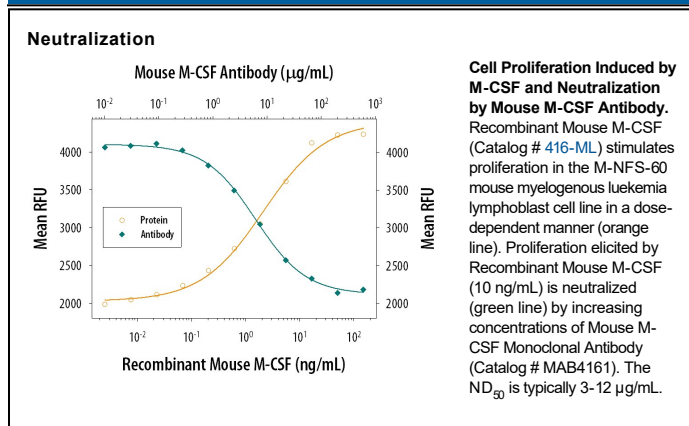
Species Reactivity	Mouse
Specificity	Detects mouse M-CSF in direct ELISAs and Western blots. In direct ELISAs and Western blots, no cross-reactivity with recombinant human M-CSF or recombinant mouse SCF is observed.
Source	Monoclonal Rat IgG _{2B} Clone # 131614
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant mouse M-CSF Lys33-Glu262 (predicted) Accession # P07141
Endotoxin Level	<0.10 EU per 1 µg of the antibody by the LAL method.
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 either lyophilized or 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	1 µg/mL	Recombinant Mouse M-CSF (Catalog # 416-ML)
Neutralization	Measured by its ability to neutralize M-CSF-induced proliferation in the M-NFS-60 mouse myelogenous leukemia lymphoblast cell line. Halenbeck, R. <i>et al.</i> (1989) <i>Biotechnology</i> 7:710. The Neutralization Dose (ND ₅₀) is typically 3-12 µg/mL in the presence of 10 ng/mL Recombinant Mouse M-CSF.	

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 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. <ul style="list-style-type: none"> ● 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.

BACKGROUND

M-CSF, also known as CSF-1, is a four- α -helical-bundle cytokine that is the primary regulator of macrophage survival, proliferation and differentiation (1-3). M-CSF is also essential for the survival and proliferation of osteoclast progenitors (1, 4). M-CSF also primes and enhances macrophage killing of tumor cells and microorganisms, regulates the release of cytokines and other inflammatory modulators from macrophages, and stimulates pinocytosis (2, 3). M-CSF increases during pregnancy to support implantation and growth of the decidua and placenta (5). Sources of M-CSF include fibroblasts, activated macrophages, endometrial secretory epithelium, bone marrow stromal cells, and activated endothelial cells (1-5). The M-CSF receptor (*c-fms*) transduces its pleiotropic effects and mediates its endocytosis. M-CSF mRNAs of various sizes occur (3-9). Full length mouse M-CSF transcripts encode a 520 amino acid (aa) type I transmembrane (TM) protein with a 462 aa extracellular region, a 21 aa TM domain, and a 37 aa cytoplasmic tail that forms a 140 kDa covalent dimer. Differential processing produces two proteolytically cleaved, secreted dimers. One is an N- and O-glycosylated 86 kDa dimer, while the other is modified by both glycosylation and chondroitin-sulfate proteoglycan (PG) to generate a 200 kDa subunit. Although PG-modified M-CSF can circulate, it may be immobilized by attachment to type V collagen (8). Shorter transcripts encode M-CSF that lacks cleavage and PG sites and produces an N-glycosylated 68 kDa TM dimer and a slowly produced 44 kDa secreted dimer (7). Although forms may vary in activity and half-life, all contain the N-terminal 150 aa portion that is necessary and sufficient for interaction with the M-CSF receptor (10, 11). The first 229 aa of mature mouse M-CSF shares 87%, 83%, 82%, and 81% aa identity with corresponding regions of rat, dog, cow, and human M-CSF, respectively (12, 13). Human M-CSF is active in the mouse, but mouse M-CSF is reported to be species-specific.

References:

1. Pixley, F.J. and E.R. Stanley (2004) Trends Cell Biol. **14**:628.
2. Chitu, V. and E.R. Stanley (2006) Curr. Opin. Immunol. **18**:39.
3. Fixe, P. and V. Praloran (1997) Eur. Cytokine Netw. **8**:125.
4. Ryan, G.R. *et al.* (2001) Blood **98**:74.
5. Makrigiannakis, A. *et al.* (2006) Trends Endocrinol. Metab. **17**:178.
6. Nandi, S. *et al.* (2006) Blood **107**:786.
7. Rettenmier, C.W. and M.F. Roussel (1988) Mol. Cell Biol. **8**:5026.
8. Suzu, S. *et al.* (1992) J. Biol. Chem. **267**:16812.
9. Manos, M.M. (1988) Mol. Cell. Biol. **8**:5035.
10. Koths, K. (1997) Mol. Reprod. Dev. **46**:31.
11. Jang, M-H. *et al.* (2006) J. Immunol. **177**:4055.
12. DeLamarter, J.F. *et al.* (1987) Nucleic Acids Res. **15**:2389.
13. Ladner, M.B. *et al.* (1988) Proc. Natl. Acad. Sci. USA **85**:6706.