

DESCRIPTION

Source *E. coli*-derived
Glu33-Ser190, with an N-terminal Met
Accession # NP_757350

N-terminal Sequence Analysis Met

Structure / Form Disulfide-linked homodimer

Predicted Molecular Mass 18.5 kDa (monomer)

SPECIFICATIONS

SDS-PAGE 37 kDa, non-reducing conditions

Activity Measured in a cell proliferation assay using M-NFS-60 mouse myelogenous leukemia lymphoblast cells. Halenbeck, R. *et al.* (1989) *Biotechnology* 7:710.
The ED₅₀ for this effect is typically 0.5-1.5 ng/mL.

Endotoxin Level <1.0 EU per 1 µg of the protein by the LAL method.

Purity >97%, by SDS-PAGE under reducing conditions and visualized by silver stain.

Formulation Lyophilized from a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution Reconstitute at 50 µg/mL in sterile PBS containing at least 0.1% human or bovine serum albumin.

Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

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.
- 3 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 human M-CSF transcripts encode a 522 amino acid (aa) type I transmembrane (TM) protein with a 464 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 223 aa of mature human M-CSF shares 88%, 86%, 81% and 74% aa identity with corresponding regions of dog, cow, mouse and rat 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. Kawasaki, E.S. *et al.* (1985) *Science* 230: 291.
13. Wong, G.G. *et al.* (1987) *Science* 235:1504.