Recombinant Human M-CSF

Catalog Number: 216-MC

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

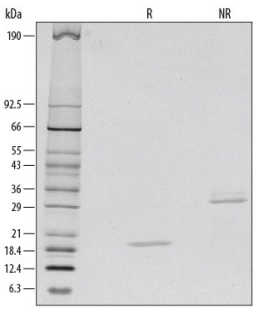
SDS-PAGE	37 kDa, non-reducing conditions
Activity	Measured in a cell proliferation assay using M-NFS-60 mouse myelogenous leukemia lymphoblast cells. Nakoinz, I. *et al.* (1990) J. Immunol. **145**:860. The ED_{50} for this effect is typically 0.5-1.5 ng/mL. The specific activity of Recombinant Human M-CSF is approximately 1.68 x 10^5 IU/µg, which is calibrated against recombinant human M-CSF WHO International Standard (NIBSC code: 89/512).
Endotoxin Level	<1.0 EU per 1 µg of the protein by the LAL method.
Purity	>97%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
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 50-500 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.

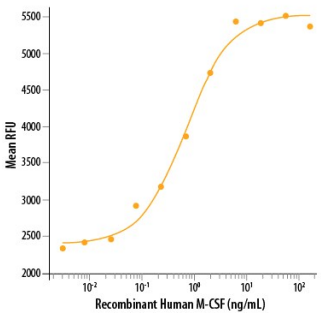
DATA

SDS-PAGE



1 µg/lane of Recombinant Human M-CSF was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by silver staining, showing bands at 19 kDa and 35 kDa, respectively.

Bioactivity



Recombinant Human M-CSF (Catalog # 216-MC) stimulates cell proliferation of the M-NFS-60 mouse myelogenous leukemia lymphoblast cell line in a dose-dependent manner. The ED_{50} for this effect is typically 0.5-1.5 ng/mL.

bio-techne
bio-techne.com
Global info@bio-techne.com techsupport@bio-techne.com
North America TEL 800 343 7475 Europe | Middle East | Africa TEL +44 (0)1235 529449
China info.cn@bio-techne.com TEL +86 (21) 52380373
Rest of World bio-techne.com/find-us/distributors TEL +1 612 379 2956

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 pleotropic 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:

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bio-techne.com
Global info@bio-techne.com techsupport@bio-techne.com
North America TEL 800 343 7475 Europe | Middle East | Africa TEL +44 (0)1235 529449
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