

**DESCRIPTION**

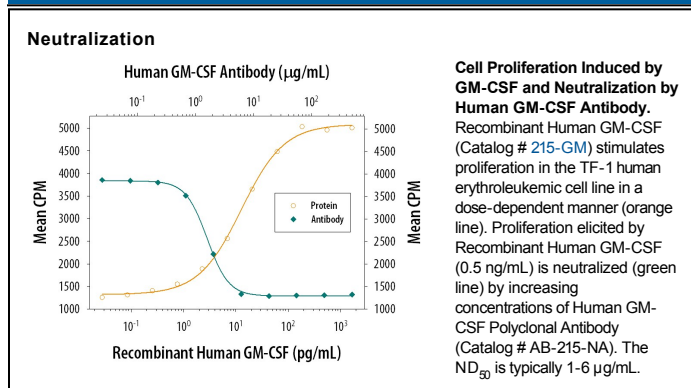
<b>Species Reactivity</b>	Human
<b>Specificity</b>	Detects human GM-CSF in direct ELISAs and Western blots. In direct ELISAs, no cross-reactivity with recombinant human (rh) G-CSF, rhIL-1 $\alpha$ , rhIL-1 $\beta$ , rhIL-2, rhIL-3, rhIL-4, rhIL-6, rhTNF- $\alpha$ or rhTNF- $\beta$ is observed. Neutralizes the biological activity of both rhGM-CSF and natural human GM-CSF. It will not neutralize the biological activity of recombinant mouse GM-CSF.
<b>Source</b>	Polyclonal Goat IgG
<b>Purification</b>	Protein A or G purified
<b>Immunogen</b>	<i>E. coli</i> -derived recombinant human GM-CSF Ala18-Glu144 Accession # P04141
<b>Endotoxin Level</b>	<0.10 EU per 1 $\mu$ g of the antibody by the LAL method.
<b>Formulation</b>	Lyophilized from a 0.2 $\mu$ m filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

**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.

	<b>Recommended Concentration</b>	<b>Sample</b>
<b>Western Blot</b>	1 $\mu$ g/mL	Recombinant Human GM-CSF (Catalog # 215-GM)
<b>Neutralization</b>		Measured by its ability to neutralize GM-CSF-induced proliferation in the TF-1 human erythroleukemic cell line. Kitamura, T. <i>et al.</i> (1989) <i>J. Cell Physiol.</i> <b>140</b> :323. The Neutralization Dose (ND <sub>50</sub> ) is typically 1-6 $\mu$ g/mL in the presence of 0.5 ng/mL Recombinant Human GM-CSF.

**DATA**



**PREPARATION AND STORAGE**

<b>Reconstitution</b>	Reconstitute at 1 mg/mL in sterile PBS.
<b>Shipping</b>	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
<b>Stability &amp; Storage</b>	<b>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</b> <ul style="list-style-type: none"> <li>● 12 months from date of receipt, -20 to -70 °C as supplied.</li> <li>● 1 month, 2 to 8 °C under sterile conditions after reconstitution.</li> <li>● 6 months, -20 to -70 °C under sterile conditions after reconstitution.</li> </ul>

**BACKGROUND**

GM-CSF was initially characterized as a factor that can support the *in vitro* colony formation of granulocyte-macrophage progenitors. It is also a growth factor for erythroid, megakaryocyte, and eosinophil progenitors. GM-CSF is produced by a number of different cell types (including T cells, B cells, macrophages, mast cells, endothelial cells, fibroblasts, and adipocytes) in response to cytokine or inflammatory stimuli. On mature hematopoietic cells, GM-CSF is a survival factor for and activates the effector functions of granulocytes, monocytes/macrophages, and eosinophils (1, 2). GM-CSF promotes a Th1 biased immune response, angiogenesis, allergic inflammation, and the development of autoimmunity (3-5). It shows clinical effectiveness in ameliorating chemotherapy-induced neutropenia, and GM-CSF transfected tumor cells are utilized as cancer vaccines (6, 7). The 22 kDa glycosylated GM-CSF, similar to IL-3 and IL-5, is a cytokine with a core of four bundled  $\alpha$ -helices (8-12). Mature human GM-CSF shares 63%-70% amino acid sequence identity with canine, feline, porcine, and rat GM-CSF and 54% with mouse GM-CSF. GM-CSF exerts its biological effects through a heterodimeric receptor complex composed of GM-CSF R $\alpha$ /CD116 and the signal transducing common  $\beta$  chain (CD131) which is also a component of the high-affinity receptors for IL-3 and IL-5 (13, 14). In addition, GM-CSF binds a naturally occurring soluble form of GM-CSF R $\alpha$  (15). Human GM-CSF is active on canine and feline cells but not on murine cells (16-18).

**References:**

1. Martinez-Moczygamba, M. and D.P. Huston (2003) *J. Allergy Clin. Immunol.* **112**:653.
2. Barreda, D.R. *et al.* (2004) *Dev. Comp. Immunol.* **28**:509.
3. Eksioglu, E.A. *et al.* (2007) *Exp. Hematol.* **35**:1163.
4. Cao, Y. (2007) *J. Clin. Invest.* **117**:2362.
5. Fleetwood, A.J. *et al.* (2005) *Crit. Rev. Immunol.* **25**:405.
6. Heuser, M. *et al.* (2007) *Semin. Hematol.* **44**:148.
7. Hege, K.M. *et al.* (2006) *Int. Rev. Immunol.* **25**:321.
8. Kaushansky, K. *et al.* (1992) *Biochemistry* **31**:1881.
9. Diederichs, K. *et al.* (1991) *Science* **254**:1779.
10. Cantrell, M.A. *et al.* (1985) *Proc. Natl. Acad. Sci.* **82**:6250.
11. Lee, F. *et al.* (1985) *Proc. Natl. Acad. Sci.* **82**:4360.
12. Wong, G.G. *et al.* (1985) *Science* **228**:810.
13. Onetto-Pothier, N. *et al.* (1990) *Blood* **75**:59.
14. Hayashida, K. *et al.* (1990) *Proc. Natl. Acad. Sci.* **87**:9655.
15. Pelley, J.L. *et al.* (2007) *Exp. Hematol.* **35**:1483.
16. Hogge, G.S. *et al.* (1990) *Cancer Gene Ther.* **6**:26.
17. Sprague, W.S. *et al.* (2005) *J. Comp. Pathol.* **133**:136.
18. Shanafelt, A.B. *et al.* (1991) *J. Biol. Chem.* **266**:13804.