

DESCRIPTION

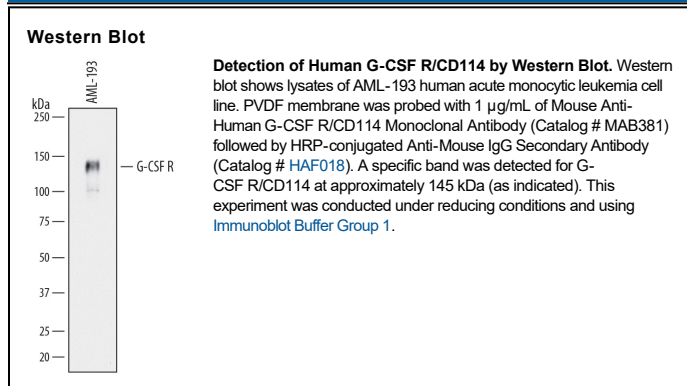
Species Reactivity	Human
Specificity	Detects human G-CSF R/CD114 in direct ELISAs and Western blots. In direct ELISAs and Western blots, no cross-reactivity with recombinant human (rh) GM-CSF R α , rhGM-CSF R β , or rhM-CSF R is observed.
Source	Monoclonal Mouse IgG ₁ Clone # 38660
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant human G-CSF R/CD114 Glu25-Pro621 Accession # Q99062
Formulation	Lyophilized from a 0.2 μ m 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 μ m 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 μ g/mL	See Below
Flow Cytometry	2.5 μ g/10 ⁶ cells	Human whole blood monocytes and granulocytes
CyTOF-ready	Ready to be labeled using established conjugation methods. No BSA or other carrier proteins that could interfere with conjugation.	

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

Granulocyte Colony Stimulating Factor (G-CSF) is a pleiotropic cytokine best known for its specific effects on the proliferation, differentiation, and activation of hematopoietic cells of the neutrophilic granulocyte lineage. G-CSF plays an important role in defense against infection, in inflammation and repair, and in the maintenance of steady state hematopoiesis. Recombinant human G-CSF has been approved for the amelioration of chemotherapy induced neutropenia as well as for severe chronic neutropenia following marrow transplant.

Cell activation by G-CSF is mediated by a type I membrane protein belonging to the cytokine receptor superfamily. Human G-CSF R, also known as colony-stimulating factor 3 receptor (CSF3R) and designated CD114, is 863 amino acids (aa) in length, with a 604 aa extracellular domain, a 26 aa transmembrane domain, and a 183 aa cytoplasmic domain that include a 23 amino acid signal sequence. As a result of alternative splicing, at least four isoforms of G-CSF R that differ in their C-terminal region exist. Isoform 2 lacks the transmembrane region and may represent a soluble form of the receptor; however the existence of soluble G-CSF R in human serum has not been reported (1). Mutations have been found in the gene encoding G-CSF R in some patients with severe congenital neutropenia. These mutations typically led to a truncation in the cytoplasmic domain of the G-CSF R leading to maturation arrest of neutrophil precursors in the bone marrow and neutropenia in peripheral blood (2). Human and mouse G-CSF R have a homology of 62.5%.

G-CSF R is expressed in mature neutrophils, neutrophilic precursors, myeloid leukemia cells, and placenta. Binding of G-CSF to its receptor induces dimerization or oligomerization of the receptor activating cytoplasmic tyrosine kinases. Signal transduction from pathways that involve Janus tyrosine kinases/signal transducer and activator of transcription proteins (Jak1, Jak2, and Tyk2/STAT3, STAT3, and STAT6), src-related protein tyrosine kinases (Lyn and Syk), Ras/MAP kinase, and phosphatidylinositol have been reported to be activated upon G-CSF stimulation (1).

References:

1. Nicola, N.A., in *Cytokine Reference*, (2001) Oppenheim, J.J. and M. Feldmann, eds. Academic Press p. 1935
2. Mitsui, T. *et. al.* (2003) *Blood*. **101**:2990.