

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse G-CSF R/CD114 in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant human (rh) G-CSF R, rhGM-CSF R beta, recombinant mouse (rm) GM-CSF R alpha, or rmM-CSF R is observed.
Source	Monoclonal Rat IgG _{2B} Clone # 680206
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived recombinant mouse G-CSF R/CD114 Cys26-Asp626 Accession # P40223
Conjugate	Alexa Fluor 405 Excitation Wavelength: 405 nm Emission Wavelength: 421 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

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.

Neutralization Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

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

Stability & Storage Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

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 and granulocyte lineage (1). G-CSF plays an important role in defense against infection, in inflammation and repair, and in the maintenance of steady state hematopoiesis. Cell activation by G-CSF is mediated by granulocyte colony stimulating factor receptor alpha (G-CSF R; also CD114), a 95-105 kDa type I transmembrane protein and member of the cytokine receptor superfamily, type I cytokine receptor family, and type 2 subfamily of receptor proteins. Mouse G-CSF R is synthesized as an 837 amino acid (aa) precursor that contains a 25 aa signal sequence, a 601 aa extracellular domain (ECD), a 24 aa transmembrane region, and a 187 aa cytoplasmic tail. The ECD contains one Ig-like C2-type domain, five fibronectin type-III domains, and 11 potential sites for N-linked glycosylation. Within the ECD there is also a WSXWS motif (aa 319-323) that is necessary for proper protein folding and thereby efficient intracellular transport and cell-surface receptor binding (2). Also, within the cytoplasmic domain there is a Box 1 motif which is required for JAK interaction and/or activation (1). Mouse G-CSF R shares 63% aa sequence identity with human G-CSF R. G-CSF R is expressed in mature neutrophils, neutrophilic precursors, myeloid leukemia cells, and placenta (1). Mutations have been found in the gene encoding G-CSF R in some patients with severe congenital neutropenia (1). These mutations typically lead to a truncation in the cytoplasmic domain of the G-CSF R leading to maturation arrest of neutrophilic precursors in the bone marrow and neutropenia in peripheral blood (3). Binding of G-CSF to its receptor induces dimerization or oligomerization of the receptor activating cytoplasmic tyrosine kinases (2). Signal transduction from pathways that involve Janus tyrosine kinases/signal transducer and activator of transcription proteins (Jak1, Jak2, and Tyk2/STAT3 and STATG), src-related protein tyrosine kinases (Lyn and Syk), Ras/MAP kinase, and phosphatidylinositol have been reported to be activated upon G-CSF stimulation (4).

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