Mouse G-CSF R/CD114
Alexa Fluor® 350-conjugated Antibody

Monoclonal Rat IgG_{2A} Clone # 723806

Catalog Number: FAB60391U

100 µg

DESCRIPTION

Species Reactivity	Mouse
Specificity	Detects mouse G-CSF R/CD114 in direct ELISAs. In direct ELISAs, no cross-reactivity with recombinant mouse (rm) GM-CSF R alpha, rmM-CSF R, recombinant human (rh) G-CSF R alpha, or rhGM-CSF R beta is observed.
Source	Monoclonal Rat IgG_{2A} Clone # 723806
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 350 Excitation Wavelength: 346 nm Emission Wavelength: 442 nm
Formulation	Supplied 0.2 mg/mL in a saline solution containing BSA and Sodium Azide. See Certificate of Analysis for details. *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.*

	Recommended Concentration	Sample
Flow Cytometry	0.25-1 µg/10^6 cells	Mouse splenocytes

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).

References:

1. Ward, A.C. (2007) Front. Biosci. **12**:608.
2. Layton, J.E. and N.E. Hall (2006) Front. Biosci. **11**:3181.
3. Mitsui, T. *et al.* (2003) Blood **101**:2990.
4. Nicola, N.A. in *Cytokine Reference*, 2001, Oppenheim, J.J. and M. Feldmann, eds. Academic Press p.1935.

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