

**Mouse Csf1r Antibody (C-term) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP13911b****Specification**

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**Mouse Csf1r Antibody (C-term) Blocking peptide - Product Information**Primary Accession [P09581](#)**Mouse Csf1r Antibody (C-term) Blocking peptide - Additional Information****Gene ID** 12978**Other Names**

Macrophage colony-stimulating factor 1 receptor, CSF-1 receptor, CSF-1-R, CSF-1R, M-CSF-R, Proto-oncogene c-Fms, CD115, Csf1r, Csfmr, Fms

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody AP13911b was selected from the C-term region of Mouse Csf1r. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

**Format**

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

**Storage**

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

**Precautions**

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

**Mouse Csf1r Antibody (C-term) Blocking peptide - Protein Information****Name** Csf1r**Synonyms** Csfmr, Fms**Function**

Tyrosine-protein kinase that acts as a cell-surface receptor for CSF1 and IL34 and plays an essential role in the regulation of survival, proliferation and differentiation of hematopoietic precursor cells, especially mononuclear phagocytes, such as macrophages and monocytes. Promotes the release of pro-inflammatory chemokines in response to IL34 and CSF1, and thereby plays an important role in innate immunity and in inflammatory processes. Plays an important role in the regulation of osteoclast proliferation and differentiation, the regulation of bone resorption, and is required for normal bone and tooth development. Required for normal male and female fertility, and for normal development of milk ducts and acinar structures in the mammary gland during pregnancy. Promotes reorganization of the actin cytoskeleton, regulates formation of membrane ruffles, cell adhesion and cell migration, and promotes cancer cell invasion. Activates

several signaling pathways in response to ligand binding, including the ERK1/2 and the JNK pathway (By similarity). Phosphorylates PIK3R1, PLCG2, GRB2, SLA2 and CBL. Activation of PLCG2 leads to the production of the cellular signaling molecules diacylglycerol and inositol 1,4,5-trisphosphate, that then lead to the activation of protein kinase C family members, especially PRKCD. Phosphorylation of PIK3R1, the regulatory subunit of phosphatidylinositol 3-kinase, leads to activation of the AKT1 signaling pathway. Activated CSF1R also mediates activation of the MAP kinases MAPK1/ERK2 and/or MAPK3/ERK1, and of the SRC family kinases SRC, FYN and YES1. Activated CSF1R transmits signals both via proteins that directly interact with phosphorylated tyrosine residues in its intracellular domain, or via adapter proteins, such as GRB2. Promotes activation of STAT family members STAT3, STAT5A and/or STAT5B. Promotes tyrosine phosphorylation of SHC1 and INPP5D/SHIP-1. Receptor signaling is down-regulated by protein phosphatases, such as INPP5D/SHIP-1, that dephosphorylate the receptor and its downstream effectors, and by rapid internalization of the activated receptor. In the central nervous system, may play a role in the development of microglia macrophages (By similarity).

**Cellular Location**

Cell membrane; Single-pass type I membrane protein. Note=The autophosphorylated receptor is ubiquitinated and internalized, leading to its degradation

**Tissue Location**

Widely expressed..

**Mouse Csf1r Antibody (C-term) Blocking peptide - Protocols**

Provided below are standard protocols that you may find useful for product applications.

- [Blocking Peptides](#)

**Mouse Csf1r Antibody (C-term) Blocking peptide - Images****Mouse Csf1r Antibody (C-term) Blocking peptide - Background**

Csf1r is a protein tyrosine-kinase transmembrane receptor for CSF1 and IL34.

**Mouse Csf1r Antibody (C-term) Blocking peptide - References**

Gueller, S., et al. J. Leukoc. Biol. 88(4):699-706(2010)Nagamachi, A., et al. Dev. Biol. 345(2):226-236(2010)Wei, S., et al. J. Leukoc. Biol. 88(3):495-505(2010)Maitra, R., et al. J. Immunol. 185(3):1485-1491(2010)Aikawa, Y., et al. Nat. Med. 16(5):580-585(2010)