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CSF1R Antibody
Purified Mouse Monoclonal Antibody
Catalog # AO1890a

Specification

CSF1R Antibody - Product Information

Application	**E, WB, IF, IHC**
Primary Accession	P07333
Reactivity	**Human**
Host	**Mouse**
Clonality	**Monoclonal**
Isotype	**IgG2b**
Calculated MW	**108kDa KDa**

Description
The protein encoded by this gene is the receptor for colony stimulating factor 1, a cytokine which controls the production, differentiation, and function of macrophages. This receptor mediates most if not all of the biological effects of this cytokine. Ligand binding activates the receptor kinase through a process of oligomerization and transphosphorylation. The encoded protein is a tyrosine kinase transmembrane receptor and member of the CSF1/PDGF receptor family of tyrosine-protein kinases. Mutations in this gene have been associated with a predisposition to myeloid malignancy. The first intron of this gene contains a transcriptionally inactive ribosomal protein L7 processed pseudogene oriented in the opposite direction.

Immunogen
Purified recombinant fragment of human CSF1R (AA: 344-497) expressed in E. Coli.

Formulation
Purified antibody in PBS with 0.05% sodium azide

CSF1R Antibody - Additional Information

Gene ID 1436

Other Names
Macrophage colony-stimulating factor 1 receptor, CSF-1 receptor, CSF-1-R, CSF-1R, M-CSF-R, 2.7.10.1, Proto-oncogene c-Fms, CD115, CSF1R, FMS

Dilution
E~~1/10000
WB~~1/500 - 1/2000
IF~~1/200 - 1/1000
IHC~~1/200 - 1/1000

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions
CSF1R Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

CSF1R Antibody - Protein Information

Name CSF1R

Synonyms 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 (PubMed:20504948, PubMed:30982609). 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 (PubMed:30982608).

Cellular Location
Cell membrane; Single-pass type I membrane protein

Tissue Location
Expressed in bone marrow and in differentiated blood mononuclear cells

CSF1R Antibody - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

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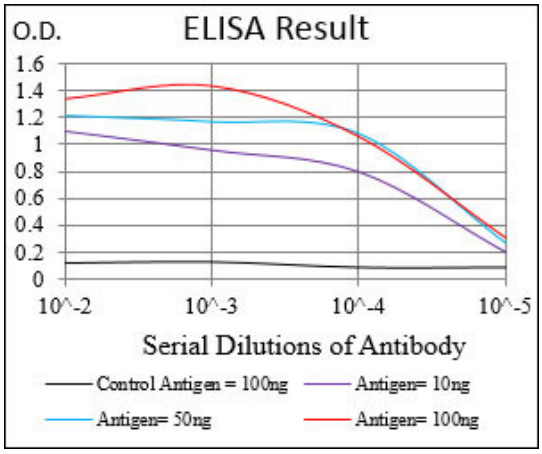

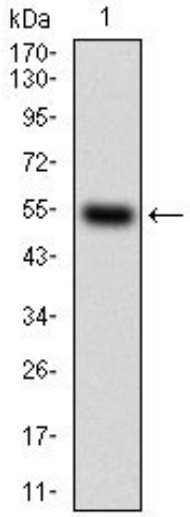

Figure 1: Western blot analysis using CSF1R mAb against human CSF1R (AA: 344-497) recombinant protein. (Expected MW is 43.3 kDa)

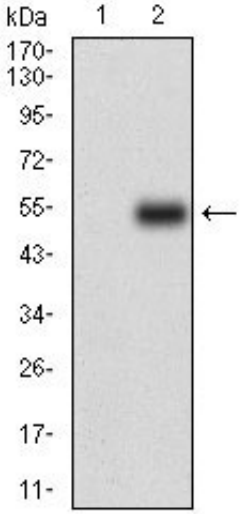

Figure 2: Western blot analysis using CSF1R mAb against HEK293 (1) and CSF1R (AA: 344-497)-hIgGFc transfected HEK293 (2) cell lysate.

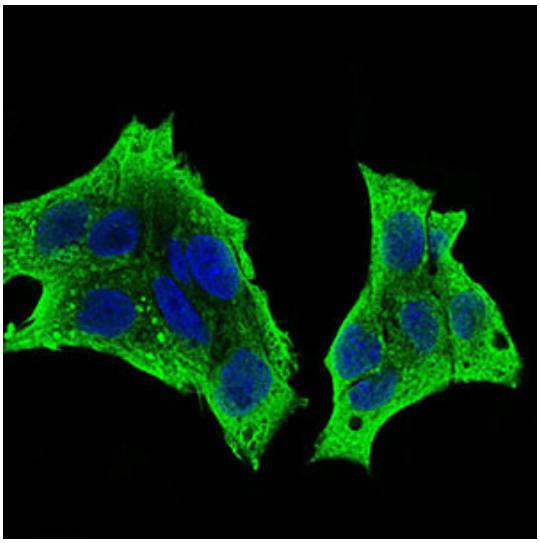

Figure 3: Immunofluorescence analysis of HepG2 cells using CSF1R mouse mAb (green). Blue: DRAQ5 fluorescent DNA dye. Secondary antibody from Fisher (Cat#: 35503)

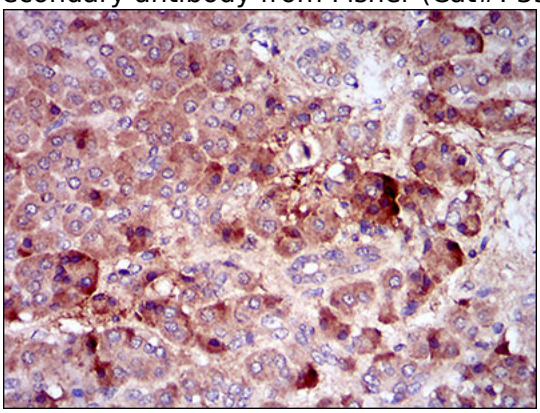

Figure 4: Immunohistochemical analysis of paraffin-embedded pancreas tissues using CSF1R mouse mAb with DAB staining.

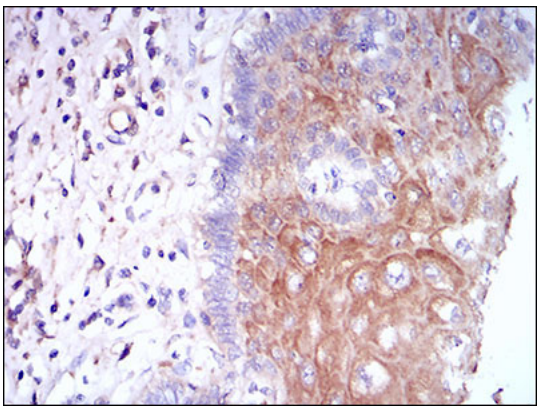

Figure 5: Immunohistochemical analysis of paraffin-embedded esophageal tissues using CSF1R mouse mAb with DAB staining.

CSF1R Antibody - Background

The membrane-associated protein encoded by this gene is a member of the superfamily of ATP-binding cassette (ABC) transporters. ABC proteins transport various molecules across extra- and intra-cellular membranes. ABC genes are divided into seven distinct subfamilies (ABC1, MDR/TAP, MRP, ALD, OABP, GCN20, White). This protein is a member of the MDR/TAP subfamily. Members of the MDR/TAP subfamily are involved in multidrug resistance. The protein encoded by this gene is an

ATP-dependent drug efflux pump for xenobiotic compounds with broad substrate specificity. It is responsible for decreased drug accumulation in multidrug-resistant cells and often mediates the development of resistance to anticancer drugs. This protein also functions as a transporter in the blood-brain barrier. ;

CSF1R Antibody - References

1. PLoS One. 2011;6(11):e27450. 2. J Biochem. 2012 Jan;151(1):47-55.