

CSFR (Phospho-Tyr561) Antibody

Purified Rabbit Polyclonal Antibody (Pab) Catalog # AP52419

Specification

CSFR (Phospho-Tyr561) Antibody - Product Information

Application WB, IHC
Primary Accession P07333

Reactivity Human, Mouse, Rat

Host Rabbit
Clonality Polyclonal
Calculated MW 107984

CSFR (Phospho-Tyr561) 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, Proto-oncogene c-Fms, CD115, CSF1R, FMS

Dilution

WB~~1:1000 IHC~~1:50~100

Format

Rabbit IgG in phosphate buffered saline (without Mg2+ and Ca2+), pH 7.4, 150mM NaCl, 0.09% (W/V) sodium azide and 50% glycerol.

Storage Conditions

-20°C

CSFR (Phospho-Tyr561) 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

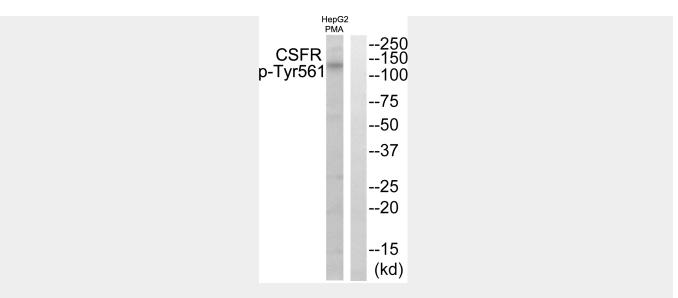
CSFR (Phospho-Tyr561) Antibody - Protocols

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

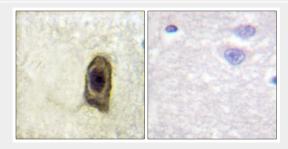
- Western Blot
- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- <u>Immunofluorescence</u>
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

CSFR (Phospho-Tyr561) Antibody - Images





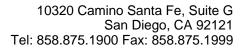
Western blot analysis of extracts from HepG2 cells treated with PMA, using CSFR (Phospho-Tyr561) antibody.



Immunohistochemistry analysis of paraffin-embedded human brain, using CSFR (Phospho-Tyr561) antibody.

CSFR (Phospho-Tyr561) Antibody - Background

Tyrosine-protein kinase that acts as 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 proinflammatory 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. 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.





CSFR (Phospho-Tyr561) Antibody - References

Hampe A.,et al.Oncogene Res. 4:9-17(1989). Coussens L.,et al.Nature 320:277-280(1986). Andre C.,et al.Genomics 39:216-226(1997). Jin P.,et al.Arthritis Res. Ther. 10:R73-R73(2008). Schmutz J.,et al.Nature 431:268-274(2004).