SANTA CRUZ BIOTECHNOLOGY, INC.

c-Fms/CSF-1R (C-20): sc-692

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

c-Fms/CSF-1R, also designated macrophage colony-stimulating factor receptor (M-CSFR), FIM2 or CD115, is a transmembrane tyrosine kinase receptor belonging to the CSF1/PDGF receptor family. It is encoded by the c-Fms proto-oncogene and is expressed in mononuclear phagocytes, oocytes, decidual cells, trophoblastic cells and some myoblasts. It is important for growth and differentiation of myeloid cells and its function can be regulated by SLAP-2. c-Fms/CSF-1R is responsible for mediating all of the functions of M-CSF. M-CSF is a glycoprotein required for the proliferation and differentiation of mononuclear phagocytes, including osteoclasts. M-CSF has also been identified as an important mediator of the inflammatory response and can regulate the release of proinflammatory cytokines from macrophages.

CHROMOSOMAL LOCATION

Genetic locus: CSF1R (human) mapping to 5q32; Csf1r (mouse) mapping to 18 E1.

SOURCE

c-Fms/CSF-1R (C-20) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of c-Fms/CSF-1R of human origin.

PRODUCT

Each vial contains 100 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-692 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

c-Fms/CSF-1R (C-20) is recommended for detection of c-Fms/CSF-1R of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

c-Fms/CSF-1R (C-20) is also recommended for detection of c-Fms/CSF-1R in additional species, including equine, canine, bovine, feline and porcine.

Suitable for use as control antibody for c-Fms/CSF-1R siRNA (h): sc-29220, c-Fms/CSF-1R siRNA (m): sc-29847, c-Fms/CSF-1R shRNA Plasmid (h): sc-29220-SH, c-Fms/CSF-1R shRNA Plasmid (m): sc-29847-SH, c-Fms/CSF-1R shRNA (h) Lentiviral Particles: sc-29220-V and c-Fms/CSF-1R shRNA (m) Lentiviral Particles: sc-29847-V.

Molecular Weight of unprocessed c-Fms/CSF-1R: 130 kDa.

Molecular Weight of processed c-Fms/CSF-1R: 165 kDa.

Positive Controls: RAW 264.7 whole cell lysate: sc-2211, THP-1 cell lysate: sc-2238 or HL-60 whole cell lysate: sc-2209.

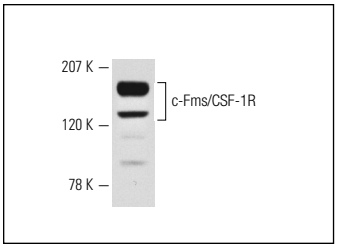
STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

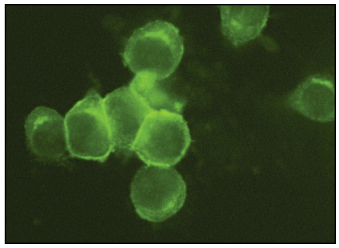
RESEARCH USE

For research use only, not for use in diagnostic procedures.

DATA



c-Fms/CSF-1R (C-20): sc-692. Western blot analysis of c-Fms/CSF-1R expression in RAW 264.7 whole cell lysate. The two major bands represent differentially glycosylated receptors.



c-Fms/CSF-1R (C-20): sc-692. Immunofluorescence staining of methanol-fixed RAW 264.7 cells showing membrane staining.

SELECT PRODUCT CITATIONS

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2. Hiyoshi, M., et al. 2008. Interaction between Hck and HIV-1 Nef negatively regulates cell surface expression of M-CSF receptor. Blood 111: 243-250.
3. Bourgin-Hierle, C., et al. 2008. Src-family kinases play an essential role in differentiation signaling downstream of macrophage colony-stimulating factor receptors mediating persistent phosphorylation of phospholipase C-γ2 and MAP kinases ERK1 and ERK2. Leukemia 22: 161-169.
4. Menke, J., et al. 2009. CSF-1 signals directly to renal tubular epithelial cells to mediate repair in mice. J. Clin. Invest. 119: 2330-2342.
5. Deng, Y.Y., et al. 2010. Microglia-derived macrophage colony stimulating factor promotes generation of proinflammatory cytokines by astrocytes in the periventricular white matter in the hypoxic neonatal brain. Brain Pathol. 20: 909-925.
6. Leung, R., et al. 2010. Filamin A regulates monocyte migration through Rho small GTPases during osteoclastogenesis. J. Bone Miner. Res. 25: 1077-1091.
7. Baek, J.H., et al. 2015. IL-34 mediates acute kidney injury and worsens subsequent chronic kidney disease. J. Clin. Invest. 125: 3198-3214.
8. Iwatake, M., et al. 2015. Punicalagin attenuates osteoclast differentiation by impairing NFATc1 expression and blocking Akt- and JNK-dependent pathways. Mol. Cell. Biochem. 407: 161-172.