

c-Fgr (N-47): sc-130

BACKGROUND

Src is the human homolog of the v-Src gene of the rous sarcoma virus, also designated avian sarcoma virus or ASV. Src was the first proto-oncogenic non-receptor tyrosine kinase characterized in human. The Src family, which has common structural motifs, is composed of nine members in vertebrates, including Src, Yes, Fgr, Frk, Fyn, Lyn, Hck, Lck and Blk. Src-family kinases transduce signals that are involved in the control of a variety of cellular processes, including proliferation, differentiation, motility and adhesion. Src-family kinases contain an amino-terminal cell membrane anchor followed by an SH3 domain and an SH2 domain, which are involved in modular association and activation, respectively. Src-family kinases, which are normally maintained in an inactive state and can be activated transiently during cellular events such as mitosis. Different subcellular localizations of Src-family kinases may be important for the regulation of specific cellular processes such as mitogenesis, cytoskeletal organization and membrane trafficking. c-Fgr is a human non-receptor tyrosine kinase family member that was discovered by using a probe toward the v-Fgr portion of the cell-derived domain of Gardner-Rasheed feline sarcoma virus. The human c-Fgr gene encodes a 529 amino acid protein.

CHROMOSOMAL LOCATION

Genetic locus: FGR (human) mapping to 1p36.11.

SOURCE

c-Fgr (N-47) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the N-terminus of c-Fgr of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-130 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

c-Fgr (N-47) is recommended for detection of c-Fgr p55 of 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).

Suitable for use as control antibody for c-Fgr siRNA (h): sc-39229, c-Fgr shRNA Plasmid (h): sc-39229-SH and c-Fgr shRNA (h) Lentiviral Particles: sc-39229-V.

Molecular Weight of c-Fgr: 55 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200.

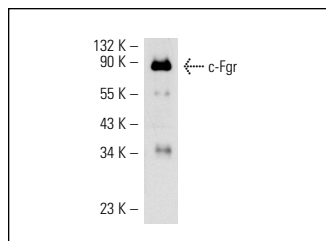
STORAGE

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-Fgr (N-47): sc-130. Western blot analysis of a human recombinant c-Fgr fusion protein.

SELECT PRODUCT CITATIONS

1. Brunati, A.M., et al. 1993. Isolation and identification of two proto-oncogene products related to c-Fgr and fyn in a tyrosine-protein-kinase fraction of rat spleen. *Eur. J. Biochem.* 216: 323-327.
2. Nijhuis, E., et al. 2002. Src kinases regulate PKB activation and modulate cytokine and chemoattractant-controlled neutrophil functioning. *J. Leukoc. Biol.* 71: 115-124.
3. Kasper, B., et al. 2003. Platelet factor 4 (PF-4)-induced neutrophil adhesion is controlled by Src-kinases while PF-4-mediated exocytosis requires the additional activation of p38 MAP kinase and phosphatidylinositol 3-kinase. *Blood* 103: 1602-1610.
4. Rollet-Labelle, E., et al. 2004. Recruitment of the cross-linked opsonic receptor CD32A (FcγRIIA) to high-density detergent-resistant membrane domains in human neutrophils. *Biochem. J.* 381: 919-928.
5. Ottonello, L., et al. 2005. CCL3 (MIP-1α) induces *in vitro* migration of GM-CSF-primed human neutrophils via CCR5-dependent activation of ERK 1/2. *Cell. Signal.* 17: 355-363.
6. Mallozzi, C., et al. 2005. Protein phosphatase 1α is tyrosine-phosphorylated and inactivated by peroxynitrite in erythrocytes through the src family kinase Fgr. *Free Radic. Biol. Med.* 38: 1625-1636.
7. Ostapkowicz, A., et al. 2006. Lipid rafts remodeling in estrogen receptor-negative breast cancer is reversed by histone deacetylase inhibitor. *Mol. Cancer Ther.* 5: 238-245.
8. Tibaldi, E., et al. 2011. Interaction between the SH3 domain of Src family kinases and HTLV-1 p13's proline rich motif: a novel mechanism underlying delivery of Src family kinases to mitochondria. *Biochem. J.* 439: 505-516.

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