SANTA CRUZ BIOTECHNOLOGY, INC.

FGFR-3 (H-100): sc-9007

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Acidic and basic fibroblast growth factors (FGFs) are members of a family of multifunctional polypeptide growth factors that stimulate proliferation of cells of mesenchymal, epithelial and neuroectodermal origin. Like other growth factors, FGFs act by binding and activating specific cell surface receptors. These include the Flg receptor or FGFR-1, the Bek receptor or FGFR-2, FGFR-3, FGFR-4, FGFR-5 and FGFR-6. These receptors usually contain an extracellular ligand-binding region containing three immunoglobulin-like domains, a transmembrane domain and a cytoplasmic tyrosine kinase domain. The gene encoding human FGFR-3 maps to chromosome 4p16.3 and is alternatively spliced to produce three isoforms that are expressed in brain, kidney and testis. Defects in FGFR-3 are associated with several diseases, including Crouzon syndrome, achondroplasia, thanatophoric dysplasia, craniosynostosis adelaide type and hypochondroplasia. Mutations in FGFR-3 are also a cause of some bladder and cervical cancers.

CHROMOSOMAL LOCATION

Genetic locus: FGFR3 (human) mapping to 4p16.3; Fgfr3 (mouse) mapping to 5 B2.

SOURCE

FGFR-3 (H-100) is a rabbit polyclonal antibody raised against amino acids 25-124 of FGFR-3 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.

APPLICATIONS

FGFR-3 (H-100) is recommended for detection of FGFR-3 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).

Suitable for use as control antibody for FGFR-3 siRNA (h): sc-29314, FGFR-3 siRNA (m): sc-35367, FGFR-3 shRNA Plasmid (h): sc-29314-SH, FGFR-3 shRNA Plasmid (m): sc-35367-SH, FGFR-3 shRNA (h) Lentiviral Particles: sc-29314-V and FGFR-3 shRNA (m) Lentiviral Particles: sc-35367-V.

Molecular Weight of non-glycosylated FGFR-3: 97 kDa.

Molecular Weight of mature FGFR-3: 135 kDa.

Molecular Weight of FGFR-3 precursor: 125 kDa.

Positive Controls: K-562 whole cell lysate: sc-2203, T-47D cell lysate: sc-2293 or Hep G2 cell lysate: sc-2227.

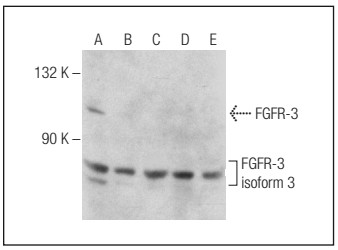
STORAGE

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



FGFR-3 (H-100): sc-9007. Western blot analysis of FGFR-3 expression in K-562 (**A**), MCF7 (**B**), T-47D (**C**) and Hep G2 (**D**) whole cell lysates and human liver tissue extract (**E**).

SELECT PRODUCT CITATIONS

1. Chesi, M., et al. 2001. Activated fibroblast growth factor receptor 3 is an oncogene that contributes to tumor progression in multiple myeloma. Blood 97: 729-736.
2. Kirby, J.L., et al. 2003. Characterization of fibroblast growth factor receptors expressed in principal cells in the initial segment of the rat epididymis. Biol. Reprod. 68: 2314-2321.
3. Meyer, A.N., et al. 2004. The cytoplasmic tyrosine kinase PYK2 as a novel effector of fibroblast growth factor receptor 3 activation. J. Biol. Chem. 279: 28450-28457.
4. Trudel, S., et al. 2005. CHIR-258, a novel, multitargeted tyrosine kinase inhibitor for the potential treatment of t(4;14) multiple myeloma. Blood 105: 2841-2948.
5. Bisping, G., et al. 2006. Targeting receptor kinases by a novel indolinone derivative in multiple myeloma: abrogation of stroma-derived interleukin-6 secretion and induction of apoptosis in cytogenetically defined subgroups. Blood 107: 2079-2089.
6. Trudel, S., et al. 2006. The inhibitory anti-FGFR-3 antibody, PRO-001, is cytotoxic to t(4;14) multiple myeloma cells. Blood 107: 7039-4046.
7. He, L., et al. 2010. Physical basis behind achondroplasia, the most common form of human dwarfism. J. Biol. Chem. 285: 30103-30114.
8. Chen, F., et al. 2011. The A391E mutation enhances FGFR3 activation in the absence of ligand. Biochim. Biophys. Acta 1808: 2045-2050.
9. He, L., et al. 2011. FGFR3 heterodimerization in achondroplasia, the most common form of human dwarfism. J. Biol. Chem. 286: 13272-13281.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.