SANTA CRUZ BIOTECHNOLOGY, INC.

Brk (C-17): sc-916

BACKGROUND

Tyrosine protein kinases play crucial roles in cell proliferation, survival, adhesion and motility by regulating ligand-mediated signal transduction, cell-cycle progression and cytoskeleton function. Tyrosine kinases may also bring about the transformation of malignant cells. Breast tumor kinase, Brk (also known as PTK6), along with its murine homolog, Sik (Src-related intestinal kinase) is one such kinase. Brk is a member of a distinct family of intracellular tyrosine kinases thought to be related to the Src family of tumor-related kinases. Brk exhibits the features of a novel non-receptor tyrosine kinase, including N-terminal SH3 and SH2 domains. Brk is specifically expressed in epithelial tissues and is restricted to cell layers immediately above the proliferative cell zone in skin and alimentary canal lining. Expression of Brk in normal tissues is relatively restricted with the highest mRNA levels found in colon, small intestine and prostate. Brk is strongly expressed in many breast carcinomas but not in normal breast tissue. Brk protein is also capable of autophosphorylation, which may play a role in its regulation.

REFERENCES

1. Wilks, A.F. 1989. Two putative protein-tyrosine kinases identified by application of the polymerase chain reaction. Proc. Natl. Acad. Sci. USA 86: 1603-1607.
2. Lee, S.T., et al. 1993. A survey of protein tyrosine kinase mRNAs expressed in normal human melanocytes. Oncogene 8: 3403-3410.

CHROMOSOMAL LOCATION

Genetic locus: Ptk6 (mouse) mapping to 2 H4.

SOURCE

Brk (C-17) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of Brk of mouse 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-916 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

Brk (C-17) is recommended for detection of Brk of mouse and rat 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), immunohistochemistry (including paraffin-embedded sections) (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 Brk siRNA (m): sc-38940, Brk shRNA Plasmid (m): sc-38940-SH and Brk shRNA (m) Lentiviral Particles: sc-38940-V.

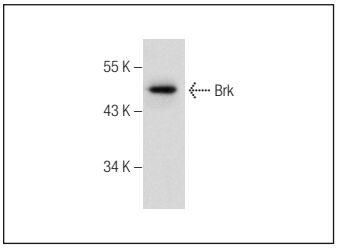
Molecular Weight of Brk: 50 kDa.

Positive Controls: mouse colon extract: sc-364238.

STORAGE

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

DATA



Brk (C-17): sc-916. Western blot analysis of Brk expression in mouse colon tissue extract.

SELECT PRODUCT CITATIONS

1. Vasioukhin, V., et al. 1997. A role for the epithelial-cell-specific tyrosine kinase Sik during keratinocyte differentiation. Proc. Natl. Acad. Sci. USA 94: 14477-14482.
2. Llor, X., et al. 1999. BRK/Sik expression in the gastrointestinal tract and in colon tumors. Clin. Cancer Res. 5: 1767-1777.
3. Derry, J.J., et al. 2000. Sik (Brk) phosphorylates Sam 68 in the nucleus and negatively regulates its RNA binding ability. Mol. Cell. Biol. 20: 6114-6126.
4. Derry, J.J., et al. 2003. Altered localization and activity of the intracellular tyrosine kinase Brk/Sik in prostate tumor cells. Oncogene 22: 4212-4220.
5. Lin, H.S., et al. 2004. Identification of tyrosine kinases overexpressed in head and neck cancer. Arch. Otolaryngol. Head Neck Surg. 130: 311-316.
6. Haegebarth, A., et al. 2004. The nuclear tyrosine kinase BRK/Sik phosphorylates and inhibits the RNA-binding activities of the Sam 68-like mammalian proteins SLM-1 and SLM-2. J. Biol. Chem. 279: 54398-54404.
7. Lukong, K.E., et al. 2005. Tyrosine phosphorylation of Sam 68 by breast tumor kinase regulates intranuclear localization and cell cycle progression. J. Biol. Chem. 280: 38639-38647.
8. Lukong, K.E., et al. 2008. Breast tumor kinase Brk, requires kinesin-2 subunit KAP3A in modulation of cell migration. Cell. Signal. 20: 432-442.
9. Lukong, K.E., et al. 2009. Brk phosphorylates PSF promoting its cytoplasmic localization and cell cycle arrest. Cell. Signal. 21: 1415-1422.
10. Miah, S., et al. 2012. Constitutive activation of breast tumor kinase accelerates cell migration and tumor growth *in vivo*. Oncogenesis 1: e11.
11. Miah, S., et al. 2014. BRK targets Dok1 for ubiquitin-mediated proteasomal degradation to promote cell proliferation and migration. PLoS ONE 9: e87684.

RESEARCH USE

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